Transactions

of the

Forty-Second Annual Meeting

of the

American Academy of Ophthalmology and Otolaryngology

CHICAGO, ILLINOIS

October 10 to 15, 1937

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1. All scientific papers and major scientific addresses
2. In Memoriam
3. Listing of Instructional Courses
4. Listing of Motion Pictures and New Instruments

BULLETIN

Minutes of Scientific Section ..... December Issue

Minutes of Business Meeting ..... December Issue
Inclusive of New Members Elected

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Printed by
Douglas Printing Company, Omaha, Nebraska

PRINTED IN THE UNITED STATES OF AMERICA

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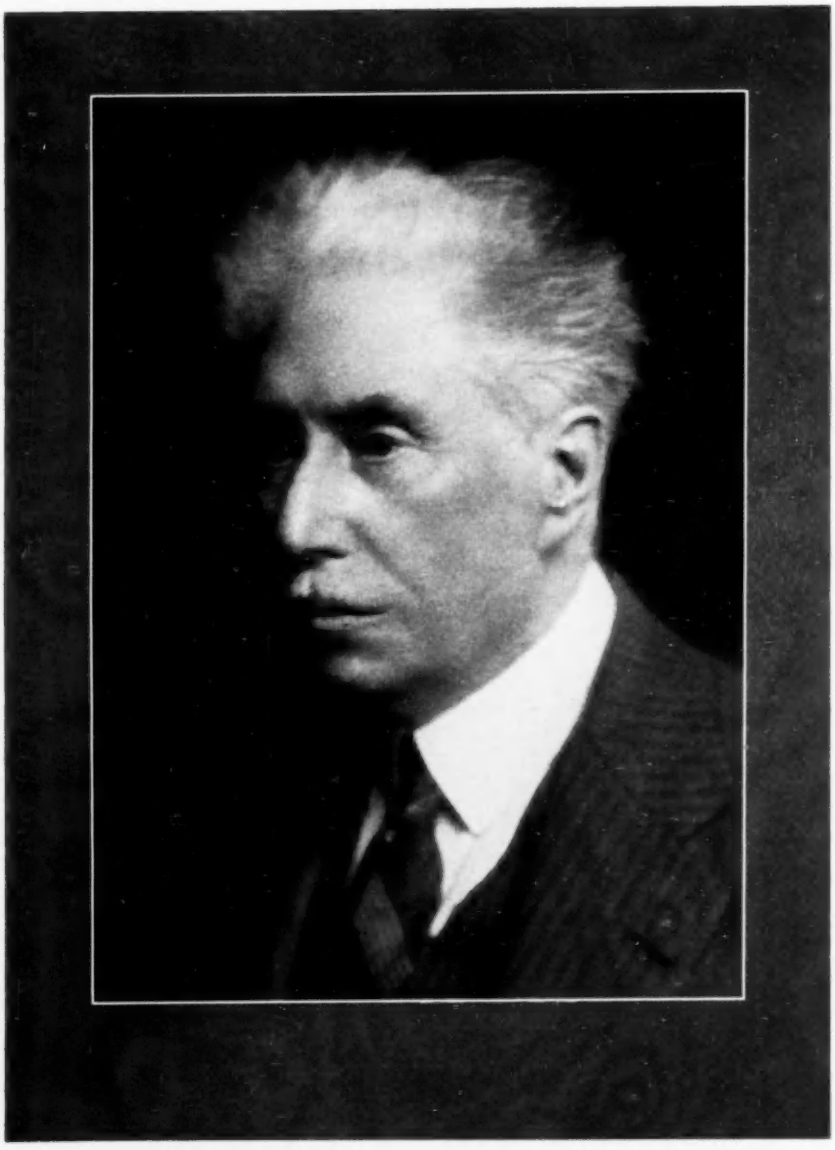

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THIS VOLUME OF THE
TRANSACTIONS OF THE AMERICAN ACADEMY OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY
IS GRATEFULLY DEDICATED TO

F. Park Lewis

M.D., F.A.C.S.

BUFFALO, NEW YORK

DOCTOR LEWIS has been a member and a sincere worker in the Academy since 1904, and was its vice-president in 1911. Of the many outstanding achievements of this eminent scientist, perhaps the most noteworthy was the foundation of the New York State Society for the Prevention of Blindness, which group later became the nucleus of the National Society for the Prevention of Blindness. Doctor Lewis has been president of this organization since its beginning.

Doctor Lewis was born in Hamilton, Ontario, in 1855. His preparation for the special field of Ophthalmology was made at the New York Ophthalmic Institute under Herman Knapp; later, in the University of Vienna under Arlt-Eduard Jaeger, and at Moorfield's in London. He is an extensive writer and contributor to many standard periodicals, text-books, and encyclopedias.

He also holds membership in:

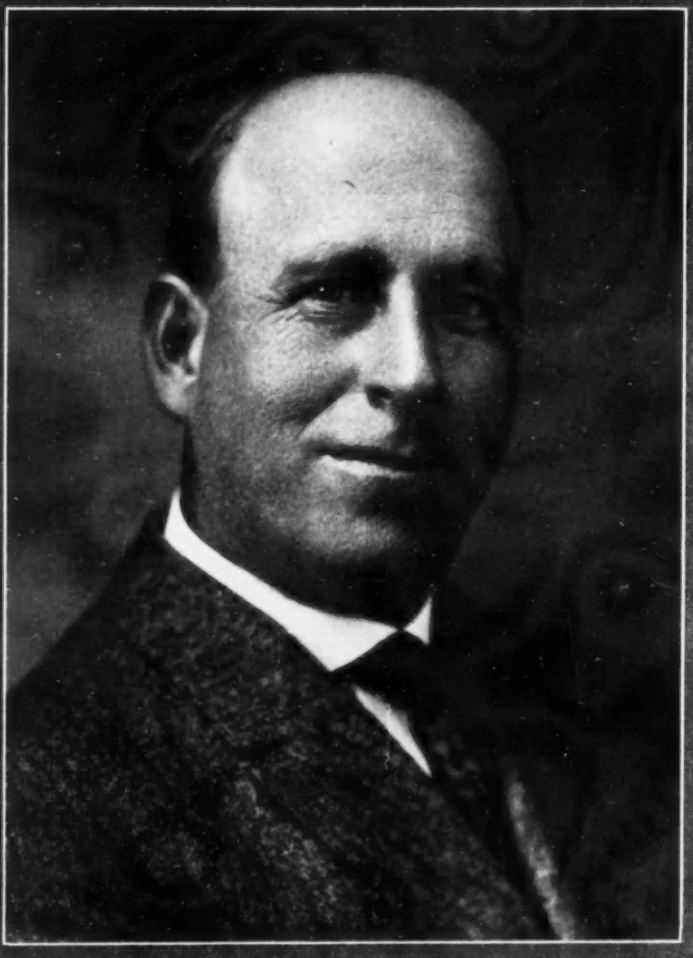
The American College of Surgeons (Fellow)
The American Medical Association
The American Ophthalmological Society
The Oxford Congress
Der Deutschen Ophthalmologischen Gesellschaft
La Société Française d'Ophtalmologie

For forty years he has been President of the Board of Visitors of the New York State School for the Blind, Batavia, New York.

Among further richly-merited honors, he was the recipient of:

The Dana medial for work for the prevention of blindness, in 1928

The Chancellor's Medal, University of Buffalo, for distinctive civic services, in 1933.



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1936—	Dr. Frank E. Burch St. Paul, Minn.	Dr. Gordon F. Harkness Davenport, Iowa	Dr. S. H. Large Cleveland, Ohio	Dr. William P. Wherry Omaha, Nebr.	New York
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The Guest of Honor

HARRIS PEYTON MOSHER, M.D.

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Joint Session

The American Academy
of Ophthalmology and Otolaryngology

Forty-Second Annual Meeting
Chicago • 1937

Address of the President

GRADUATE TEACHING

LEE W. DEAN, M.D.
SAINT LOUIS

THE RECORDS of the Academy do not show when I became a member. The first scientific paper that I presented before a national organization was presented before this group in 1900. The meeting was held in a parlor of a hotel, about twenty-two by twenty-four, and it was not overcrowded. I remember that Gradle, Casey Wood, Alt and Vail were there. I remember this well, because they treated my contribution kindly. The paper dealt with eugenics, heredity, embryology and pathology. The contribution shows that we were interested in the basic sciences. During the forty years I have been a member of this body, I have been constantly interested in and have experimented with various methods of graduate teaching of our specialty. With your permission, I desire to offer my conclusions on this subject.

Graduate education is not new. Edward Jackson in his presidential address before this body in 1904 stated that "The specialist requires primarily a special education." With his usual good judgment he spoke of a knowledge of anatomy and physiology of the eye and of general medicine as being the prime requisites for an ophthalmologist. Similarly situated now, I will be satisfied if I can arouse in some of you the idea of thinking more about this theme and help in the ultimate solution. The Teachers' Section of this Academy and the two examining boards need your help and advice. These are the agencies which have the greatest influence in molding graduate teaching of our specialty.

I have always been situated where I could carry out graduate teaching as I deemed best. There were no standing rules to follow or regulations by which to be guided. I have been influenced by what Mosher, Coates, Politzer and others have done. There are two things in which I believe: First, that if we require genuine

research of a student as a requirement for admission to the specialty we will lose to the profession many excellent clinicians. Ellett, in his presidential address before this body, said "Research is a hard problem. It may surprise you to know that the difficulty is not to finance it, but to find men capable of doing it." Individuals with the initiative necessary to do original work are scarce. An acceptable thesis treating on some scientific subject can perhaps be required. Second, that by adding years of study you cannot make a good specialist out of one who has not the requisite mental equipment. The first requirement is to choose the right man for the work. I would say that natural ability, the desire to make progress, and honesty are the three things I desire most in my graduate students. There are plenty of examples in this audience that good specialists can be made by two years of graduate training. The attendance at the educational courses at these meetings is conclusive proof that the members of this Academy still desire to make progress.

We are all proud of the record of the Academy. As an educational organization for its members, it ranks above any other medical society. Weber in his presidential address in 1910 said, "The future of the Academy depends upon maintaining a membership of the best which the profession affords." This can only be done by advancing education in our specialty more rapidly than in others. I think we will all agree that at this time graduate teaching of ophthalmology and otolaryngology is done better than in any other specialty.

As a student I was interested in the graduate teaching of ophthalmology and otolaryngology in the nineties (1890-1900). The difficulties of getting proper instruction at that time were the same as today. Everyone had trouble in securing training in the basic sciences. There was more opportunity for clinical work. At that time it was axiomatic that one should be a good doctor before becoming a specialist. Some of us who never entered general practice devoted a year to graduate study of general medicine before beginning the study of the specialty. The opportunities for instruction in ophthalmology were far better than those in otolaryngology. Ophthalmological clinics had existed for a long time, while the first two ear clinics, those of Gruber and Politzer, were still young. In the United States there was little opportunity for study in the west, middle west, or south. Most work was done in the clinics of New York or Philadelphia or in Europe. Most of the otolaryngological diseases and many of the eye at that time were treated by

the general practitioner. Many cities of 8,000 inhabitants did not have an eye, ear, nose or throat specialist. Comparatively speaking, much more time was given to the specialties in the under-graduate curriculum than today. Now diseases of the eye, ear, nose and throat are treated by ophthalmologists and otolaryngologists. This makes the graduate teaching of the specialties of equal importance with the under-graduate teaching of general surgery and medicine. In the nineties each student made his own plans for studying the basic sciences. In either specialty one could utilize the laboratories of anatomy, physiology and pathology of his own medical school and work out the various problems himself. At Moorefields, in London, the clinic of Fuchs, and that of Schnable in Vienna, courses were offered in the histology and pathology of the eye. A very few were given instruction in anatomy and pathology of the ear by the father of otology, Politzer, at his home. In the private clinic of Hajek anatomy of the nose was taught. A good way to get anatomy of the ear, nose and throat was in the anatomical institutes of Vienna or Berlin and the Cook School of Anatomy in London. No courses in histology or pathology of the nose, pharynx and larynx were offered at any place. By going into the autopsy room in the pathological institute at Vienna one could get material and give it to a diener for sectioning and staining. Then it was necessary to employ a general pathologist by the hour to help with the study of the slides. It was an advantage to have the special pathology taught by one who was a good general pathologist.

The difficulties in securing training in the basic sciences were great at that time but not insurmountable. Certainly with all our good medical schools and excellent teachers and wonderful special hospitals today, in spite of the great demand for this work, we can, if we face problems squarely, meet all the exigencies of the situation. In the nineties many ophthalmologists and otolaryngologists were not satisfied with their training. After entering practice they soon found that they lacked knowledge, particularly in certain basic sciences. They did not have access to such wonderful review courses as are given by this Academy. Nevertheless, advantage was taken of the opportunities that were at hand and many men became very well trained.

In visiting England in later years I found otolaryngologists of my age had prepared themselves in the same laboratories and in the same way as the men of this country. So far as I know there were no residencies in otolaryngology either in England or the United States at that time. During the latter part of the last cen-

tury many of the doctors in this country secured their training by entering the offices of specialists and after serving apprenticeship from two to three or more years, at least in some instances, became most excellent ophthalmologists and otolaryngologists. In some offices a certain amount of the basic sciences of ophthalmology was taught. The men who have been trained in our service have been asked about the defects in our teaching methods. The usual reply has been that they needed to know more about the technique of office practice. The development of the art of ophthalmology and otolaryngology as well as the science must be given proper consideration in our present day graduate work.

Early in this century, graduate teaching of our specialty developed rapidly in this country. A number of outstanding ophthalmologists and otolaryngologists became interested and devoted much of their time to it. Residencies of the various better types appeared here and there in the United States. Post-graduate schools developed short intensive courses and long comprehensive courses. All of these things exerted a beneficial influence on the practice of ophthalmology and otolaryngology. A great step forward was taken when in 1920 the University of Pennsylvania offered eight-month graduate courses in ophthalmology and in otolaryngology. The time was devoted mostly to the study of the basic sciences.

Early in the century there was no compulsory standard for the practice of ophthalmology or otolaryngology. There was no way of differentiating between the excellently trained and the poorly trained specialist. Any doctor could on any day without any preparation change his sign from physician and surgeon to that of eye, ear, nose and throat specialist, and then he was in fact a physician practicing in this specialty. Fortunately, the National Board of Ophthalmology in 1915 and the National Board of Otolaryngology in 1924 entered the picture and at least definitely determined the qualifications of physicians as specialists in ophthalmology and otolaryngology who were certified by either board. These two groups have been the most important factors in the recent development of the graduate teaching of these specialties. While it is ridiculous for one to study anatomy and pathology just to pass examinations, the examination is a necessity if a proper standard is to be maintained. Unconsciously, directors of various graduate residencies and of graduate teaching have been influenced by the requirements of these boards. Other specialties followed the lead of ophthalmology and otolaryngology and many examining boards were formed. An advisory board was created to coordinate the

work of the various boards. The advisory board and the Council on Medical Education and Hospitals of the American Medical Association are instituting measures for the standardization of the graduate teaching of these specialties.

I am of the opinion that a specialist should be just as well prepared for the practice in a small community as in a large city. He will receive little help in the smaller place. His patients' welfare will often rest in his hands alone. The author remembers well his early days of practice in a city of eight thousand, his first mastoid with a brain abscess, the second with a thrombophlebitis of sigmoid sinus. Good roads and the automobile have changed things. Nevertheless, the emergencies exist in small towns as well as large cities and all should be prepared to handle them as they arise. Every specialist from now on should be well prepared before he is allowed to enter the practice of his specialty or become a member of this Academy. It will not be long before the profession is overcrowded. Ethics in the medical profession have not changed in my lifetime. They have always been determined by the type of individual. If we want ethical specialists we must begin with honest men.

It is essential that many men remain eye, ear, nose and throat men. A rhinologist must have a good knowledge of ophthalmology and an ophthalmologist must be conversant with rhinology. Both must be good internists. It is axiomatic that in the great rural communities of this country the people expect one physician to care for their eyes, ears, noses and throats. The same is true of the smaller cities both in the north and south. In the large cities it is not necessary for one to cover all four fields. When the separation exists a very large percentage of our patients must see both an eye specialist and the ear, nose and throat man. If one is interested in pursuing this matter further, he should read the presidential address of Otto J. Stein before the Academy in 1909. Certainly every one of us should encompass the whole field of ophthalmology and otolaryngology, including bronchoscopy, or limit himself to one or more subjects as he thinks the exigencies of the situation demand. Each one is the best judge of what he should do. Too much regulation and regimentation is not a good thing. Certainly if one is trained in ophthalmology and otolaryngology and later gives up practicing one or more of these groups, he is so much better prepared for the practice of that part of the specialty with which he elects to continue. There are examples of this in the audience.

We are confronted today with the fact that the opportunity for graduate study does not meet the demand. The Teachers' Section of the Academy is working on this problem. We should all attend this meeting and be prepared to advise young men as to how they are to get training in the specialty. Any doctor with good training in the basic sciences of a specialty should have no difficulty in securing his clinical training. Such a knowledge is the only foundation upon which a proper conception of the specialty can be built. In some institutions efforts have been made to have the staff men give to the internes this knowledge of the basic sciences. This requires night work on the part of the residents with consequent deterioration of health, necessitates a great sacrifice on the part of the staff men, and the treatment of patients in the house is necessarily interfered with by class work. It is not fair to place patients under supervision of an interne who has not secured a fair knowledge of the subject of ophthalmology and otolaryngology. If he cannot see a drumhead properly, if he cannot make a good examination of the larynx, if he cannot make a good fundus examination, he should not be placed in charge of a patient. At any rate he should be as well prepared in ophthalmology and otolaryngology as general internes are in medicine and surgery before they begin their service. Many of our very best medical schools give only a minimum amount of ophthalmology and otolaryngology to the under-graduates. The main objection to requiring the basic science course is that the expense involved excludes many excellent students. This Academy and other agencies should make available scholarships for deserving students. We must see that the financial burden is such that the graduates of medicine without funds will have a chance. There is no question but that in the medical schools and hospitals of this country there are an abundance of facilities which if properly utilized will supply the necessary instruction in the basic sciences of ophthalmology and otolaryngology. I believe that the diagnosis of and the study of the practice of diseases of the eye, ear, nose and throat should be given at the same time the basic sciences are studied. Once the foundation is laid and the student is prepared for clinical work in the hospitals there will be little or no difficulty in finding abundant facilities in this country for carrying out proper clinical training in diseases of the eye, ear, nose and throat.

Address of the Guest of Honor

UNSOLVED PROBLEMS IN OTOLARYNGOLOGY

HARRIS P. MOSHER, M.D.
BOSTON

I HESITATED to accept the honor (and it is a very high honor) of speaking before you today, because I had nothing new to say. I took my perplexity to Fenton and Wherry, whom I have long relied upon for help in delicate matters. Both suggested that I speak again in the vein of a paper of mine of many years ago entitled "Unsolved Problems." They were insistent in their urging and discreet in their flattery. Wherry said, "Your long experience, and your position as Dean of Otolaryngology (of all things I hate is the title of Dean) make your opinions of value to us younger men." Wherry boasted a bit in those last three words. I yielded, of course. As the paper developed, I found myself assuming the role of the universal commentator. The paper, therefore, is a running comment on men and measures, past and present, with a few guesses as to the future.

The paper attempts to cover so much ground that it is very staccato in effect. The main topics and the order in which they are treated are as follows: conservatism, disappearing diseases, problems I have seen solved, unsolved problems, and finally a few paragraphs of sermonizing for young and old, to use the stock phrase of patent medicines. The first three topics are in the nature of an introduction.

CONSERVATISM

Things have to happen before they can be seen in perspective. If I had been called conservative twenty years ago, I should have taken it as an insult. I admit now the conservatism born of experience.

The immediate result of surgery in our field is often radically different from the final result. The itch to print is always with us. We often print too soon. I have. We report our results before the

hypnosis of the patient and the wishful reasoning of the surgeon, which accompany any operative procedure, have worn off. What I am trying to do is to acknowledge that I have become conservative on certain matters and to say it without hurting my pride. I shall not have to pay office rent much longer, and can afford to face my results, and question at times those of the other fellow.

DISAPPEARING DISEASES

In our contagious hospital the beds in the diphtheria ward are mostly empty. Epidemic sore throat from contaminated milk gets into the newspaper headlines but seldom. Papilloma of the larynx, and especially fibroma of the vault of the pharynx are seen occasionally with us now instead of constantly. Tuberculosis of the larynx gets to our clinic but seldom. The systemic disease is being conquered. We see very much less of the late effects of syphilis, gumma of the soft palate, and the sunken external nose with but little more than the tip projecting above the level of the face.

THINGS WHICH I HAVE SEEN SOLVED

Esophageal pouch at the pharyngeal end. There is an embryological background which plays a part in the formation of a pharyngeal esophageal pouch. Assymetry of the mouth of the esophagus throws the swallowing mechanism off center and results in an oblique thrust. The two stage external operation under local anesthesia makes the handling of this condition a solved problem. In our part of the country, Dr. Lahey was a pioneer in bringing about this result. There seems to be no age limit for the performance of the operation.

The Tear Sac Operation. To speak commercially, the modified tear sac operation is the easiest money in nose and throat. The operation properly done is ninety per cent successful. A few ophthalmologists are doing it. Quite a number of these progressives, however, are not doing it right. They pay no attention to the anterior ethmoid cells nor to a high deviation of the septum. Neglect on these two points is sure to vitiate the operation. The long bearded ophthalmologists, I speak both literally and metaphorically, who have not accepted the operation, have five more years to go before they get out of the Rip Van Winkle class.

Cardiospasm, or Fibrosis of the terminal portion of the esophagus. In patients suffering from so-called cardiospasm, as Dr. MacMillan and I see them, spasm plays but a minor part, if it plays

a part at all. Only two or three times a year does the esophagus of such a patient show spasm. It has been proved that the pathological condition causing the obstruction is a fibrosis of the terminal portion of the esophagus in the crural canal. It has been proved also that disease of the gall bladder and liver, especially of the gall bladder, often causes fibrosis of the terminal portion of the esophagus by transfer of infection. The anatomical configuration of the terminal portion of the esophagus invites stenosis and twists, and continued observation has shown that twist is equally important with the fibrosis.

The fluoroscopic examination of the esophagus and the dilatation of fibrosis of the terminal portion by sight have been, in my hands, a great advance over older methods of treatment. The majority of cases can be handled successfully in this way. In old cases where there is great dilatation of the esophagus with a double twist of the lower end, the esophageal wall is liable to be thin as the result of old ulcerations, so that all intra-esophageal manipulations are exceedingly dangerous. Such cases are candidates for an anastomosis between the lower end of the esophagus and the fundus of the stomach.

It has been proved that the esophagus can be infected before birth. In the adult, abundant pathological findings have shown that it can be infected in acute systemic diseases as well as in chronic. The infection can occur in the upper part of the esophagus as well as in the lower. These findings give an explanation for tubular strictures just below the cricoid. Dr. Macmillan is finding that these are common, even more common than fibrosis of the terminal portion of the esophagus. Up to now we have had no explanation for them. Years ago it was pointed out that exostoses or lipping of the lower cervical vertebræ was associated with webs in the pyriform sinus or back of the cricoid cartilage. These findings have driven the diagnosis of globus hystericus off the map. Evidence is accumulating that tuberculosis of the chest can cause marked changes in the esophageal wall without stricture.

I have had something to do with the subjects just mentioned. I expect praise for my restraint in disposing of them in three paragraphs.

Deep Cervical Abscess. It has taken twenty years to put deep pus in the neck partly on the map. I say partly because the general surgeon who should be fully alive to this matter is not and the general practitioner is even less aware of the source of the infection

and its full possibilities. A septicæmia accompanying or following a throat infection, however slight, means infection around or in the internal jugular vein. The anatomy of the neck and the finding of deep pus have been simplified. The anatomist is not helping the clinician by returning to the hair splitting and fascia splitting so dear to the academic heart. In deep pus high in the neck, the surgical bull's-eye is the pharyngo-maxillary fossa opposite the attachment of the tonsil to the superior constrictor. The surface landmark for this is the angle of the jaw or slightly above. The easiest, surest and safest approach to deep pus high in the neck is by way of the submaxillary fossa.

Laryngectomy. Laryngectomy has become the property of the laryngologist. MacKenty blazed the way in this country. Theoretically, the two stage operation should be the safer. In the hands of the seasoned operator, the one stage operation has conquered all objections — at least all that I can raise.

So much by way of introduction.

UNSOLVED PROBLEMS

I now come to my main topic, unsolved problems.

Chief among the unsolved problems in laryngology which I shall touch upon are chronic progressive infection of the nasal and pharyngeal mucous membrane; the radical frontal sinus operation; cancer of the esophagus; anaphylaxis, hay fever, and asthma; orbital abscess; osteomyelitis of the frontal bone; laryngo-fissure, and lung abscess.

In otology, the outstanding unsolved problems are chronic progressive deafness, otosclerosis, brain abscess and otitic meningitis. This is too much of a list to discuss thoroughly. What I have to give is more in the nature of headlines and comment.

Chronic progressive infection of the nasal and pharyngeal mucous membrane. He who can arrest or cure this condition will prevent or cure chronic progressive deafness. Such an achievement would be a monument to any man. Those who have been in practice fifteen years or more can easily recall cases of progressive infection of the nasal and pharyngeal mucous membrane for which your minutest search gave no cause, that is, no focus of infection, and for which no remedy was of avail. As time went by, if the patient still trusted you and came to you, the inferior turbinate was seen to shrink, the inferior meatus became slowly wider, the posterior pharyngeal wall thinner and dry. The cartilage of the

Eustachian tube at times shrunk and the Eustachian tube became narrow at the isthmus in one or both ears. There was a slow but steady loss of hearing. A biopsy of the nasal mucosa showed an increase of fibrous tissue. For years, I have felt that I have taken money from these patients under false pretenses.

The Radical Frontal Sinus Operation. The frontal sinus operation is still unsatisfactory. Ten per cent of our cases, i.e., those done at the Massachusetts Eye and Ear Infirmary, have to be reoperated. The betterment of the operation lies in the perfection of some technique for epidermatizing the enlarged nasofrontal duct or in obliterating the sinus. The latter is simple in theory but very uncertain as now practiced. At my suggestion, the operators at the Massachusetts Eye and Ear Infirmary in cases of osteomyelitis have routinely taken off both the anterior and posterior walls of the frontal sinus, have done a minimum amount of work in the anterior ethmoidal labyrinth and enlarged the nasal frontal duct but little if at all. No attempt was made to take off the floor of the sinus. So far, perhaps eight cases — this procedure has been uniformly successful. Where you are not dealing with osteomyelitis and wish to obliterate the frontal sinus, the anterior wall of the sinus should be removed first and the sepsis in the sinus allowed to clear up. When this has come about the posterior wall is removed. In other words, it should be a two stage operation.

The Ethmoidal Operation. The pendulum is swinging from the intranasal ethmoid operation to the external. I agree with the change. If your knowledge of applied anatomy justifies your doing any ethmoidal operating at all, thorough work can be done more safely by the external route. Making a fetish of a small incision does not appeal to me. However, it is a strong point in selling the operation to the patient and his family.

Orbital Abscess. In orbital abscess, the ophthalmologist should give up making repeated incisions through the lids. An external ethmoidal operation is indicated, as the origin of the infection is in the ethmoidal labyrinth, generally in the posterior cells. In most cases, a break will be found from the ethmoidal labyrinth into the orbit.

Focal Infection. The nose and throat as the source of chronic systemic infection (focal infection, in other words) has been a great disappointment to me in spite of radical operations at my hands or at the hands of others. One of my chief jobs of late years

has been the trying one of seeing over-operated sinus cases of my confreres from all parts of the country. You may have seen some of mine. In certain localities the opinion prevails that there is no such thing as over-operating in sinus cases, only incomplete operating.

When a man tells me he cures eighty per cent of his arthritic cases by a sinus operation, I doubt him. If he says fifty per cent, I still doubt. If he puts his figures at five to ten per cent, then I may believe him.

At this point, I cannot help interjecting the following two sentences. Don't operate a sinus case just because the other fellow will if you don't. Don't promise a worn-out wreck of 60 or 65 a return of youth by taking his tonsils out, or by exenterating his ethmoid.

Writing on an allied subject, and speaking of the frontal sinus especially, Tilley made the striking statement that he had followed not a few patients whom he saw some 15 or 20 years before who at that time had sinus infection which many would have called operable. The followup showed that they survived the acute flareup and had outgrown the chronic condition.

Goldsmith said at least one good thing when he stated that the American patient as compared with the purer Anglo-Saxon stock, he meant Canadian stock, of course, is more intolerant of slight bodily disturbances. Such complaints as a little nasopharyngeal catarrh are not fussed about in Canada nor in England. In these cases Goldsmith believes in philosophy, not surgery. I think he is right.

Osteomyelitis of the Frontal Bone. In this desperate complication of frontal sinusitis we now have, or perhaps I should say, I now have the very great comfort of a definite and systematic plan of operation. Owing to this, the results of our operations at the Infirmary are steadily getting better. This subject has been pretty thoroughly written up and I will not take the time to go over it again in detail. Those who are not familiar with the late literature would do well to make themselves so. I wish to remind you, however, that there are two types of osteomyelitis of the frontal bone, one, the overwhelming furious type wherein the infection spreads rapidly and a second type which advances more slowly and often leads to sequestration. The first type generally complicates an acutely infected frontal sinus — swimming cases being the worst; the second more often follows trauma. At times, it is hard to

tell which type you are dealing with. This has led to a sit-down strike on the part of some surgeons in dealing with this condition. If one happens to start off with a case or two of the second type, he shouts from the housetops, if he is built that way, that all cases are of this type and radical operation is not necessary. If only I knew how to pick out this type of case and be sure of it, I would operate conservatively. As I cannot, I prefer to follow the teachings of the pathological findings in the many bone specimens which have come to me for section; in other words, I prefer to operate radically. As suggested by Williams, the role played by anærobic bacteria is worth following up.

One more word on this subject and that is about pitting edema of the skin of the forehead. If there is pitting edema to the hairline or even two-thirds to the hairline, I still think that the frontal bone should come off to the hairline. When, however, the edema extends only half an inch or an inch above the rim of the orbit, I should content myself with the frontal sinus operation, that is, with a frontal sinus operation which includes the anterior wall. Great as the value of edema of the skin is in outlining the limit of the bone infection, judgment should be used in interpreting this sign. Recently, I saw a case with Dr. Poirier where the patient had an orbital abscess and there was edema and tenderness of the lower forehead for two inches above the upper orbital rim. After an external ethmoidal operation everything cleared up.

In X-ray plates of the frontal sinus where the sinus is moderately large laterally, it is common to find branching diploëtic veins running upward from the outer angle of the sinus. Often these can be seen to connect with the sinus. Recent studies have emphasized the diploëtic veins as the chief channels of infection. They should be carefully watched by repeated X-rays. I have seen the infection travel by such an infected vein a long distance before it localized.

Cancer of the Esophagus. I stand as helpless today before cancer of the esophagus as I did twenty-five years ago. A ray of hope, however, has appeared of late. Deep X-ray therapy has caused the tumor to disappear in a few cases but the esophagus was left thin and fibrosed so that it ruptured when the patient was given food by mouth. So far the remedy has proved as fatal as the disease. Nevertheless, it has been shown that X-ray can cause the disappearance of the cancer. In this there lies the hope, as better X-ray technique will undoubtedly be evolved. In carcinoma of the lower third of the esophagus, the thoracic surgeon with his improved

and modern technique is still our greatest hope. Within the past few years the whole esophagus has been removed in two or three cases. As yet this must rank only as a surgical stunt.

Anaphylaxis and Allergy. It was a landmark in laryngology when the reaction of mucous membrane to anaphylaxis was recognized. First, it was learned that in making a diagnosis of pansinusitis by X-ray, edematous mucous membrane caused by anaphylaxis must be ruled out. Then mucous polyps were proved to come and go with the lighting up or the subsidence of the anaphylactic symptoms. We have not by any means seen the end of this matter. I predict that those few who can keep sane on this subject will show us something worth while in the near future, especially if we can learn more about the chemistry of foods and also their action upon mucous membrane. I have a hunch that in certain cases nasopharyngeal catarrh, as it is called, is an anaphylactic reaction to foods. I have a suspicion that this is so in my case. But what a doctor thinks he proves on himself does not carry much weight.

Speaking of the nasopharynx, I would remind you of the familiar fact that the organisms responsible for air-borne diseases breed in the nasopharynx. Experiments are now in progress to show that bacteria laden air can be sterilized by ultra violet light. If this can be added to the air conditioning of our houses and hospitals, which is already going on, the unlovely condition of things today, namely, breathing the air exhaled from each other's nasopharynx will be done away with. This sounds utopian. But it is already being hinted at strongly.

Hay Fever and Asthma. In spite of the tremendous amount of work which has been done on hay fever, vasomotor rhinitis, and asthma, I do not approach a case of any one of the three with confidence. Hay fever, when due to a pollen which can be discovered, is the most satisfactory to treat. The theory of allergy is entirely logical. However, in the treatment of allergic states, in spite of the increasing literature to guide you, and in spite of all the late refinements in diagnosis and treatment, success is still largely a hit or miss affair. In evaluating the results of treatment, wishful reasoning plays a greater part than in any other of the diseases in our specialty. The patient is emotional and you cannot blame him because he has good cause, but the doctor often becomes so; otherwise, we would give up the case and quit. Temporary improvement after so many varieties of treatment is so common that you are worn out by the recurring cycle of hope and despair.

Vasomotor rhinitis will respond dramatically to massive cauterization (ionization) as it has responded in the past to fractional cauterization. I cannot help feeling that massive cauterization is worth while. With the revival of cauterization on what might be called the grand scale, an elaborate equipment was first thought necessary. It is not necessary now. A certain amount of permanent damage to the mucous membrane follows cauterization. It makes a great show when pictured on paper. Somehow I cannot bring myself to believe that the whole bodily economy is shaken or changed by it. I hesitate to disagree with our Chairman on another point, namely, that in all cases of vasomotor rhinitis you can find the cause of the allergic upset if you give time enough and have brains enough. His experience has not been mine.

What can you promise an asthma patient who also has proven sinus disease? Operation will help or cure in not over thirty per cent, that is, these are the figures on the Atlantic coast. It has been said that failure to get a cure is due to lack of thoroughness in operating. The claim also has been made that eighty to ninety per cent of cases can be cured. Ask the practitioners in neighboring cities who see the recurrences in these supposedly cured patients and the percentage comes down markedly.

If every ten years an operator would honestly — I say honestly, ugly as the word is, because there is no other word which conveys my meaning — if, to repeat, every ten years an operator would check up the ultimate result of his sinus cases or, perhaps better, make it possible for his friends to do it, we would get much needed information. We need the long range results — we are flooded with the others.

Tracheobronchitis. Tracheobronchitis, better named malignant or obstructive tracheobronchitis, sends on the average two or three cases a year to our children's ward or to the Children's Hospital, a separate institution. It is a disease of infancy and young children. The organism which causes it is almost always the staphylococcus albus. It is very fatal. In a few cases the child dies a heart death. In a case of this kind, the child is admitted to the hospital sick unto death. The respiratory embarrassment is relieved by tracheotomy but the child soon dies. The autopsy shows a tracheobronchitis with pneumonia, but there is not enough obstruction of the bronchi or trachea to cause death and the fatal outcome is assigned to toxemia. The majority of cases, however, have obstructive symptoms. The bronchoscope shows that the trachea and bronchi are lined with dirty adherent crusts which are removed with diffi-

culty and soon reform. The attempt to keep the trachea clean by a bronchoscope passed through the cords generally fails and a tracheotomy becomes necessary. There is progressive difficulty in breathing punctuated with attacks of extreme dyspnœa. Removal of the crusts tides the patient over from one crisis to another. Steam inhalations and the oxygen tent are the interim treatment. Helium bids fair to supplant oxygen. Many medicaments, mostly of an oily nature, have been dropped into the trachea in order to soften the crusts but they have little effect. In a recent case of Dr. Evans at the Infirmary, the child was on the point of death twice and was rescued by the bronchoscopic removal of the crusts through the tracheotomy opening. After the second emergency, Dr. Heine suggested dropping staphylococcus bacteriophage into the trachea in the hope of softening the crusts and perhaps preventing their reformation. This was done with the hoped-for result. The child promptly recovered. Seldom if ever have we had a more dramatic case.

Bronchiectasis. Bronchiectasis, if not too extensive and if confined to one lung, can be cured by lobectomy. This is a major advance. The endless and fruitless washings of the bronchoscopist are coming to an end.

The Punch Tracheotomy. Many years ago, I advocated the punch tracheotomy in foreign body cases in children where the lungs contained much secretion. This happens especially where a peanut has been inhaled. This type of tracheotomy does away with a tracheotomy tube for the first week and allows the trachea to be sucked out easily, or what is often better, permits the patient to be held upside down by the heels and quick drainage obtained.

Diathermy. The diathermy knife is a great aid in doing a tracheotomy in cases of late carcinoma of the larynx when the neck is much swollen and partly broken down by secondary infection.

Lymphatic Drainage. It has long been held and partly proved that there is a lymphatic connection between the sinuses, the pharynx, and the lungs. It remained for Larsell, by animal experimentation, to fully prove this. His work has just been completed but as yet has not been published.

Laryngofissure. In new growths of the larynx, where a biopsy is negative or difficult, a diagnostic laryngofissure should be done. It should be resorted to freely, especially in subglottic growths.

One precaution is necessary, namely, to put in a feeding tube for a few days in order to prevent mis-swallowing and infection of the lungs. This can be easily fatal.

Ozena, or Atrophic Rhinitis. I have had no success with vaccines in this condition. Theoretically, they should help. When we get further along in our knowledge of immunity and blood chemistry, I fully expect they will accomplish something. Meanwhile, the various operations for narrowing the nasal cavity have always seemed to me to be putting the cart before the horse. I am reminded, however, that putting the cart before the horse, or rather the cart before the man has been done successfully in China for centuries. I feel a bit guilty that I have not tried any of these procedures. In connection with this disease, I am afraid that I have held up my hands.

Lung Abscess and Tonsillectomy. Lung abscess is associated with tonsillectomy oftener than with any other operation. The adenoid operation which almost always goes with the tonsil operation was imported into America by way of Boston by Dr. Hooper, one of the early worthies in laryngology. He used the natural or the upright position for the performance of the operation. From the mechanical standpoint it is the best position. For many years I have been suspicious of it in connection with lung abscess. Finally, eight years ago, I got tired of the continued slurs about the "graveyard position" as the upright position was nicknamed in certain parts of the country, and ordered all tonsil and adenoid patients to be operated in the prone position.

SUMMARY OF THE STUDY OF TONSILLECTOMIES DONE AT THE MASSACHUSETTS EYE AND EAR INFIRMARY FROM APRIL 1, 1929, TO APRIL 1, 1937, WITH RESPECT TO LUNG ABSCESS FOLLOWING OPERATION

All figures have been divided into a group before April 1, 1933, when the Rose position of the patient during tonsillectomy was substituted for the upright position on those patients who were operated on under ether anesthesia, and a group following April 1, 1933.

Number of tonsillectomies and adenoidectomies done at the Massachusetts Eye and Ear Infirmary under ether anesthesia against number of lung abscesses developing shortly after operation in the same patients.

Group I	Tonsillectomies and Adenoidectomies	Lung Abscesses
April 1, 1929, to April 1, 1930	2347	
April 1, 1930, to April 1, 1931	2150	
April 1, 1931, to April 1, 1933	4536	
Total before the Rose operation	8853	12

Group II	Tonsillectomies and Adenoidectomies	Lung Abscesses
April 1, 1933, to April 1, 1935	4193	
April 1, 1935, to April 1, 1936	1645	
April 1, 1936, to April 1, 1937	1630	
Total with the Rose position	7468	1

During the last four years that the upright position for operating on patients for tonsillectomy under ether anesthesia was used, 12 cases of lung abscess developed shortly after operation, or 0.13 per cent of the tonsillectomies and adenoidectomies done under these circumstances developed lung abscesses. On the other hand, during the first four years that the Rose position of the patient for a similar operation was used, 1 case of lung abscess developed, or 0.013 per cent of the cases of tonsillectomy and adenoidectomy developed lung abscess. Since the Rose position for operating has been adopted, the proportion of post tonsillectomy lung abscesses has been cut to one-tenth the proportion that developed when the patient was in the sitting position during the operation. (The one case of lung abscess that developed in the second period could not be definitely proved to be a lung abscess and was thought by some of the medical consultants to be merely a pulmonary infarct without frank suppuration.)

An analysis of the etiological agents of lung abscess as presented by the histories of lung abscess cases that entered the Massachusetts General Hospital during a nine year period which includes the eight years under discussion in regards to the lung abscesses that followed tonsillectomies and adenoidectomies at the Massachusetts Eye and Ear Infirmary.*

AN ANALYSIS OF THE ETIOLOGICAL AGENTS OF LUNG ABSCESS

as presented by the histories of lung abscess cases that entered the Massachusetts General Hospital during a nine year period, which includes the eight years under discussion in regards to the lung abscesses that followed tonsillectomies and adenoidectomies at the Massachusetts Eye and Ear Infirmary.

	Number	Per cent of lung abscess
Pneumonia	141	45.0%
Tooth extraction	21	6.7%
Foreign body in the lung	10	3.2%
Laporotomies	14	4.5%
Miscellaneous surgery	12	3.8%
Tumor of the lung	3	1.0%
Puerperal	5	1.6%

Ear, Nose and Throat Surgery:	Number	Per cent of lung abscess
Sinus surgery	4	1.3%
Mastoid surgery	2	0.6%
Radical removal of nose for carcinoma	1	0.3%
Biopsy of larynx and laryngofissure	1	0.3%

* These figures were tabulated by Dr. G. G. Houston and Dr. R. G. Snow.

Tonsillectomies done elsewhere.....	87	27.8%
Tonsillectomies done at Massachusetts Eye and Ear.....	11*	3.6%
Local tonsillectomy done at Massachusetts Eye and Ear.....	1†	0.3%
Total Ear, Nose, and Throat Surgery.....	107	34.3%§

Total Lung Abscesses admitted to the Massachusetts General Hospital during the years 1929-1937, inclusive..313

Cavernous Sinus Thrombosis. There is such a thing as aseptic thrombosis of the lateral sinus. Although it is much more rare, the same condition can occur in the cavernous sinus. The usual type of infection in the cavernous sinus, however, is of the fulminating fatal type. In the literature a few cases of recovery of the fulminating type have been reported after exenteration of the orbit and enlarging the sphenoidal fissure (Mosher operation) combined with tying the common carotid artery (Eagleton operation).

Naturally, it is hard to get the patient's consent to the removal of the eye. This is the great drawback to the operation.

My feeling is that vision in the eye will be lost anyway, if not the whole eye, should the patient survive long enough. This opinion is based upon three or four autopsy specimens of cavernous sinus cases. In each the orbital fat was studded with abscesses. The infection ran along the sheath of the optic nerve to the globe of the eye, and in the last case there was a separation of the retina due to hemorrhage. In this case, also, there was a sizable abscess at the apex of the orbit next to the lateral wall of the ethmoid labyrinth.

For some years now I have had the belief that the cavernous sinus often discharges its pus into the apex of the orbit and that drainage of the sinus can in a measure be secured by doing a thorough ethmoidal exenteration with complete removal of the lamina papyracea plus an incision through Tenon's capsule into the apex of the orbit. If, as I believe, the sight is lost anyway, the operator does not have to think of the optic nerve. I have a hunch that this

* One lung abscess treated outside the Massachusetts General Hospital is included in the table of lung abscesses developing after tonsillectomy done at the Massachusetts Eye and Ear Infirmary.

† One tonsillectomy was done under local anesthesia at the same time that a submucous resection of the septum was done also under local anesthesia.

§ Although 34.3% of the lung abscesses treated at the Massachusetts General Hospital were following ear, nose and throat operations, only 6.5% of the lung abscesses treated there were following ear, nose and throat surgery at the Massachusetts Eye and Ear Infirmary.

procedure is worth a routine trial. If it works only in the occasional case, it would still be worth while.

THE TRAINING OF THE SPECIALIST

The training necessary for the practice of a specialty is very much of an unsolved problem. The various Examining Boards and the Advisory Council of the Specialties are struggling with it. The vital questions pressing for answer are, how long should the instruction in the basic sciences take? Who is to give it? and How long should the special internships be and where can they be obtained? Dr. Wherry estimates that 160 men a year should be provided for. There are not enough special hospitals to take them.

The answers to some of the above questions, as I see them, are as follows:

Four or five centers can be established about the country where basic training can now be given. The instructors are to be drafted from the medical schools, or better, men on the hospital staffs should be trained to give it. In Boston, at the Massachusetts Eye and Ear Infirmary, this is now possible. The men having taken basic courses become candidates for the House Officerships in the special hospitals as far as these positions go. How to dispose of the rest still remains the great problem. By a concerted effort other hospitals with special departments, even though they take but one or two candidates apiece, can help out very materially and it should not be long before the 160 men can be taken care of.

What worries me most is how to supply training for men who cannot afford two and a half to three years for preparation. For those men, who hope to be consultants or to obtain teaching positions, three years are none too long. However, for men who are to practice in smaller places than the larger cities which are the natural centers, two years, or even less, will make them qualified to practice otolaryngology. The best that we have to offer such men at present is a number of intensive basic courses, and a graduate assistantship in the Out-Patient Department of a special hospital. The right kind of a man can get his training in this way, especially if his general internship included surgery. Some of us, and I am one, got most of our training this way.

UNSOLVED PROBLEMS IN OTOLOGY

The Nasopharynx and the Ear. There used to be intense surgical activity in the treatment of the nasopharynx. That is, in my beginning days, the posterior pillar was stripped of thickened mu-

cous membrane and adhesions in the fossa of Rosenmueller were repeatedly broken down or curetted. The Eustachian tube was regularly dilated with bougies, especially after Yankauer perfected his instruments. What has become of these procedures? Most of us employ them but occasionally. The rise and fall of therapeutic measures is disconcerting. It is often embarrassing when you realize that all of this is taken out of the patient and the patient's pocket-book. Often, enthusiasm is checked by judgment very late.

Meningismus-Meningitis. The term meningismus or meningeal irritation has never appealed to me. The phenomenon is due to infection. It may pass off if the body is able to take care of it, and this does happen, or it may blossom into a frank meningitis. To make the diagnosis of meningismus is misleading, even dangerous, because it is an ostrich-like procedure and gives a false sense of security.

Meningitis is one of the chief, if not the chief, of unsolved otological problems. It is always hanging over the ear surgeon. Naturally, no ear condition has been attacked in so many ways. Through and through drainage of the meninges by the puncture of the lateral ventricle, the posterior cistern, or by laminectomy have not given consistent results. They are eminently logical, and their failure is very disappointing. Decreasing the intracranial pressure and depleting the amount of cerebrospinal fluid by magnesium sulphate is also a rational procedure and has given a few favorable results.

I rely on a thorough removal of the focus of infection in the mastoid with a liberal exposure of the lateral sinus and the dura. Incision of the dura is almost a routine. The operation is followed up by repeated lumbar punctures.

Cushing, some thirty years ago, damned lumbar puncture and Ayer, ten years later, after some animal experimentation, again blasted it. In the hands of the otologist it has not proved dangerous. I could not practice otology without it.

One confusing thing about meningitis is that cases occasionally get well almost of their own accord. Even a cloudy fluid with bacteria does not always spell disaster. A culture to be positive should contain organisms in both the spinal fluid and the blood stream.

In making a diagnosis of meningitis by the examination of the spinal fluid, some writers hold that the presence of 10 cells, 7 cells being taken as normal, proves meningitis. I think this is drawing the line too fine.

The injection of various dyes into the blood stream has given isolated recoveries, generally in the hands of the originator of the method. A wave of popularity for the individual drug has followed promptly the publication of the experimenter's initial success, only to be followed by the gradual abandonment of the drug. Vaccines are worth trying. They are credited with occasional successes.

Prontysil and Prontylin, which are on the crest of the wave at the moment, have the most promising record to date. In fact, the reports are really startling.

By this time, my readers have sensed that I am a firm believer in biochemistry as the most likely source of our advance in therapeutics. If the surgeon can unlock the infection, nature can work miracles through the fighting and defensive qualities of the blood. How to stimulate the blood chemistry along the right lines is the problem of the immunologist and biochemist.

Otosclerosis. Otosclerosis is still unsolved. The so-called treatment of this condition continues to be the great field of the quack. Intense work is being done on this condition. The old idea that heredity plays a part in its transmission still holds. Pre-birth bone changes, especially about the fissule ante fenestrum, are striking and it would seem as if they must play a part in the final pathological changes. Alterations in bone chemistry, especially as to the phosphates and calcium have come to nothing so far. My opinion, however, is that our hope in this disease lies in biochemistry, prenatal and postnatal. In speaking of biochemistry, we must not forget the endocrines. Why does the disease start at puberty and why is it more prevalent in females than in males? These are unsolved problems.

Labyrinthitis. Early operation before the infection has produced a pool of pus in the internal auditory meatus is essential. Some fifteen years ago when Richards of New York startled all of us with his imported labyrinthine technique, there was a great increase in labyrinth operations. As time went on, however, the operative fervor subsided. It was soon proved over again that many a chronic ear with labyrinthine symptoms lost them after a radical mastoid operation. My belief is that in many, if not in most cases, a relatively simple operation, namely, enlarging and connecting the round and the oval window and making an opening in the promontory of the cochlea, is sufficient. All otologists should master this operative technique. The dread of the labyrinth and the facial nerve is such that many have not. Nevertheless, one should

make himself as much at home in this technique as in that of the radical mastoid.

Symptoms of a suppurative labyrinth plus a rising cell count in the spinal fluid are the main signs for immediate operation. Facial paralysis shows that the infection has extended to the labyrinthine capsule. This also means immediate operation.

It has been held by Frazer and the German school that if a patient gives a history of a recent suppurative labyrinthitis and presents himself with a dead labyrinth and a chronic running ear calling for a radical mastoid, early operation in such cases is very liable to be followed by meningitis.

Brain Abscess. The problem of brain abscess is, first, to find it, and then to drain it successfully. The otologists and the neurological surgeons, at least many of them, hold opposite views about drainage. The age-old method of treating an abscess is to open it, and keep it open until it ceases to drain. The otologist acts on this principle. The wire gauze drain has a good record of recoveries, and so have the methods of strip packing, either with plain gauze or with strips protected with rubber tissue. Rubber tubing has not as good a record.

In contrast to this method of drainage, the neurological surgeon drains the abscess by repeated puncture with a trocar canula, hoping that the abscess will cease to fill and become a sterile foreign body. When this happens the end treatment is to remove the abscess capsule as if it were a tumor. To my mind each puncture of the abscess is a possible source of infection of the meninges or the cerebral tissue.

Both the otologist and the neurological surgeon agree on one point, namely, to delay exploring for a brain abscess a reasonable time in order to let it wall off. Non-encapsulated brain abscesses are hard to drain, and give a very high mortality.

Osteomyelitis of the frontal bone is a great cause of brain abscess. Such abscesses are very fatal, because they tend to be multiple and bilateral.

King pointed out that a brain abscess tends to come to the surface and if opened before this, turns wrong side out and becomes superficial.

If I should be asked to give a routine treatment of encapsulated brain abscess, it would be as follows: After exposure of the dura, wall off with diathermy a circle of dura an inch to an inch and one-half wide. In the center of this explore for the abscess with

the Cushing needle. If pus is found, hold the needle in place in the abscess, and with the diathermy needle cut out a cone of brain tissue around the needle a quarter of an inch in diameter down to the abscess and then continue through the abscess wall. Put in strip drainage or the wire gauze drain, whichever you prefer. I still like the wire gauze drain.

Unfortunately, all brain abscesses are not simple; often they are multiocular, and drainage fails whatever its character. At our hospital, I feel that we have not made full use of the encephalogram as an aid in the diagnosis of brain abscess.

The Physiology of the Ear. Due to the investigations of gifted workers, the knowledge of the physics of sound has advanced in a most awe-inspiring fashion. Since the Weber-Bray phenomenon startled and stunned us, the physiologists have charted the pathways of the auditory impulses from the internal ear through the substations of the brain to the cortex. The organ of Corti has been put on the throne again. The place where the various tones of the scale are heard has been accurately determined. The tectorial membrane is almost forgotten. Papers on this new knowledge have crowded the programs of our societies and left many of us gasping for breath, in some cases the readers of the papers, also.

The Audiometer. The audiometer has been, I will not say actually debunked, but it has settled down to an instrument of record. The high hopes which many of us had that it was to solve some of the problems of hearing have not been fulfilled. Its most important defect, namely, its inability to produce a continuous tone, is only now being remedied.

Hearing Aids. The manufacturers of one of the well-known aids to hearing now say that they can adjust their machine to the hearing defect of any given patient. A prediction that this would be accomplished eventually was made some ten years ago. It has long been the dream of the otologist. It is to be hoped that there is some basis of fact behind this announcement.

Petrositis. For the past few years petrositis has ruled the day. In French literature, there are occasional cases which go back for years, showing that the condition has been known for a long time. What has happened is that the signs and symptoms of this condition have been systematized and correlated, the pathological conditions back of them made clear, and feasible operative approaches to the petrous tip have been worked out. The profession has had

to learn the anatomy of the temporal bone as it never knew it before. Unless there is a fistula to act as a lead, there is no easy way to open the petrous tip. Frankly, I do not like burr operations. You cannot set up surveying instruments in the middle ear. When possible, surgery should be a matter of sight, not feeling. For this reason the temporal fossa approach has always appealed to me. I do not feel, however, that the final technique of this approach has been worked out.

There are one or two things about this condition of petrositis which I cannot understand, namely, it has been held that there is no anatomical connection between the mastoid cells and the cells of the petrous tip. Gross anatomical specimens seem to show that there is. The natural, I almost said the common sense, way for the cells to act is for them to continue on to the tip.

I believe infection finds no difficulty in finding a direct path from the mastoid cells to the petrous tip, or from the tip to the cells crowning the middle ear.

You may call infection of the petrous tip osteomyelitis and make much of the red bone marrow there, as Eagleton does, but from the practical standpoint, once infected, I do not believe that an infected petrous tip need be considered essentially different from an infected and broken-down mastoid tip.

Another point in these cases which has come home to me forcibly is that a poor X-ray is of no diagnostic value. The X-ray must be of the highest grade, and all three of the standard positions must be taken before any reliable judgment is possible.

I feel that Eagleton's idea of approaching the tip from the pharynx is to be taken seriously.

Paralysis of the Facial Nerve. Paralysis of the facial nerve either from disease or trauma I consider one of the nearly solved problems, due to the work of Ballance and Dench. It remains only to try out the technique already given and to evaluate it.

Ménière's Disease. Ménière's disease is in the same category as facial paralysis. It is on the way toward surgical solution. The cutting of the vestibular portion of the eighth nerve, after the technique of McKenzie, has given brilliant results. Another technique has arisen lately in Boston which is still in the experimental stage. It consists of exposure by way of the temporal fossa of the superior semicircular canal, opening the canal and destroying the vestibular labyrinth by the insertion of a cautery point.

Middle Ear Surgery. Middle ear surgery in the hands of many, and I am one of them, has fallen into disuse. It is much simpler and more direct to do a radical. The pioneers in ear surgery, that is the men of two generations ago, were very adept in middle ear surgery. Since that time, middle ear surgery, at least in Boston, has gradually been given up in favor of the radical mastoid. I feel that otology has lost a good deal in allowing middle ear surgery to slip backward and should strongly advise beginners in otology to perfect themselves in the technique of middle ear surgery for use in selected cases.

* * *

So much for unsolved problems. The concluding pages are in a different vein. From now on I am giving unasked advice. This goes with grey hairs. Recently, I read somewhere a definition of an educated man which can be applied. This is the first one of the kind which I have seen. It is as follows: "An educated man is one who thinks things through to a conclusion, whose output is steady and uniformly of good quality." In medicine, the lowest type of productivity is the verbatim case report. Uncorrelated facts are as useless as a star in the Milky Way. In dealing with young specialists, yes, even with older ones, the hard thing to find is a man who can set a problem for himself and make an honest attempt to solve it on his own initiative.

The following paragraph jumps in at this point without introduction. It is entitled the "Loneliest Moment."

The loneliest moment in a surgical career is when you face an autopsy, where, as the phrase goes, "It is on you." What was not clear in life is only too clear after death. The professional pathologist has never been responsible for a human life. His greatest delight is in finding an adequate cause of death. In discussing your patient's case, he becomes, it seems to you, almost garrulous. In it he finds a rare specimen and many good teaching points. You just have to stand there and take it.

TEACHING

The phrase "teachers are born, not made" is more than threadbare, and less than half true. The willingness to teach is what counts and the willingness to be on time and to teach to the end of the job. Financial independence is a great help to contented teaching and in some positions absolutely necessary. A robust per-

sonality also helps. Nothing gives a medical school more of a black eye than to advertise graduate courses and have them half given. Better no graduate courses at all. In teaching, give everything you have. Your sidelines may be more stimulating than your main line. Your wife can help in this matter. She can grant you the time either graciously or grudgingly. If she chooses the latter course, you sample Hell on earth.

The following few paragraphs are for the younger men.

But once before have I preached in print. That little sermon has travelled widely. I am not going to repeat it. I should, however, like to say the following as a possible addition.

Once a day go to your den and take half an hour, better an hour, and think things over. Thinking, as I understand it, is to evaluate things or happenings — it is the solving of a problem or working at one. It is not day-dreaming — not watching the Fords go by. Eastern nations have a daily period of meditation, as they call it, as a part of their religion.

Speaking of dens, you can tell a man by his den. This, however, is aside from the subject. I repeat, go to your den and think. This will put it to at least one good use, and in a measure offset the hideous taste you may have shown in furnishing it.

One more remark in the preaching vein, and I speak from experience. Keep alive some of the finer things which you practiced in your youth, — collecting books, new or old — your music — the reading of worth while books, — yes, even the reading of poetry. It takes conscious effort to do this with the radio and the automobile tempting you every minute of your waking hours.

Symposium on Physical Therapy

THE ACTION OF ROENTGEN RAYS AND RADIUM ON THE EYE AND EAR

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THE MAJORITY of human beings prize their sense of vision so highly that any impairment of it is regarded as a calamity. To preserve unimpaired vision or to recover impaired vision many persons will go to great trouble and expense. This general appreciation of the advantage of vision probably explains the efforts of the medical profession to prevent diseases of the eye and to seek improved methods of remedying such diseases or of alleviating their effects.

Fear that roentgen rays or radium might have an injurious effect on the eye, the observation of ocular disturbances following exposure of the eye to these agents, or the desire to test their possible therapeutic value in diseases of the eye, has led to numerous experimental studies on animals. As a result, our knowledge of the effects of irradiation on the ocular structures is considerable. Unfortunately, much of this information lies in experimental reports in several languages published in a diversity of medical periodicals in many different countries.

EXPERIMENTAL BACKGROUND (EYE)

During the early years of radiology, it was observed that, when the entire body of an animal was exposed to roentgen rays or radium, the eyes sometimes gave evidence that excessive exposure caused irritation or inflammation of the eyelids and conjunctiva, and sometimes also of the cornea, but these observations seldom received the attention required to determine whether the disturb-

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ances noted were a direct effect of irradiation or whether they were secondary to the action of the rays on other structures or to disturbances in general metabolism.

Von Hippel,[69, 70, 71] Bossuet, and Pagenstecher irradiated the pregnant uterus of guinea pigs, rabbits and dogs and subsequently observed developmental anomalies of the eyelids and degenerative changes in the crystalline lens of some of the fetuses. But because of inadequate controls and insufficient or ambiguous data, it is impossible to accept the evidence as conclusive. Much later, Little and Bagg observed similar anomalies after prenatal irradiation, and they apparently showed that the changes in the lids may be transmitted to the descendents through several generations. This would indicate that the effects noted are due to action of the rays on the germ plasm of the parents.

Von Hippel, who exposed the entire body of pregnant rabbits (including the abdomen), noted in some of the fetuses degenerative changes in the lens ranging from slight opacity to cataract. However, similar degeneration of the fetal lens was observed in one of the young produced by four females whose abdomen had been carefully shielded from direct irradiation. Moreover, since similar changes in the young were found after solutions of choline had been injected into mothers who had not been exposed to the rays, the ocular abnormalities must have resulted from a general toxic effect produced by irradiation or by choline. The experiments of Bossuet also are not conclusive. More thorough investigations by Chalupecký, by Tribondeau and Récamier, by Belley, and Tribondeau and Lafargue,[155] and by others, clearly showed that exposure to roentgen rays of the rapidly developing eyes of young animals (soon after birth) may retard the development of the eyeball as a whole and may induce pathologic alterations in the lids, conjunctiva, cornea, lens and iris. Retardation of development was the greater the younger the animal, and it varied in degree with the dose of rays and with the time elapsing between irradiation and observation. Retardation sufficient to cause marked difference in the size of the eyeball was a constant result of irradiating the eyes of very young animals when sufficient time was allowed for the difference to manifest itself. However, since the growth of the bones and other structures included in the field of irradiation was similarly retarded, it is not certain whether the inhibition of growth of the eyeball was a direct or indirect effect, or whether it was part of a general effect on all the tissues included in the exposed territory.

Eyelids and conjunctiva. — In many experiments in which the eyes of immature as well as adult animals were exposed to a sufficient dose of roentgen rays, the first effects noted were loss of hair and cilia and inflammation of the eyelids and conjunctiva. Actually, the conjunctiva appeared to be slightly more sensitive than the lids because the conjunctivitis usually antedated the blepharitis. From one to three weeks after irradiation of the eye, the hair and cilia became loose and could be readily extracted; they began to fall spontaneously a few days later. From a few days to a week later, the conjunctiva became red and injected, and this was accompanied by increase in secretion. The secretion was mucous in character or, when irradiation had been sufficiently intense, the secretion became mucopurulent or purulent; sometimes these disturbances continued for several weeks. At the same time, or soon afterward, the eyelids themselves became red, thickened and edematous. When the dose of rays had not been too large, the alopecia of the eyelids was only temporary, and the lashes and hair grew again later. Irradiation beyond a certain limit caused permanent alopecia, with atrophy or ulceration of the lids. Belley and Bossuet observed that the regrowing hair was white at first, but that later the pigment also returned. Irradiation sufficient to cause severe conjunctivitis also induced degeneration of the endothelial lining of the blood vessels; this was accompanied by perivascular leukocytic infiltration and was followed by regenerative hyperplasia of the endothelial cells, thickening of the vascular coats and narrowing of the lumen. Moreover, the degree of the pathologic change was proportional to the doses of roentgen rays to which the animals had been exposed. However, as Jacoby[72] contended, the epilation dose for the eyelids of the rabbit, as given by Rados and Schinz, undoubtedly was too high. It seems clearly established, therefore, that the radiosensitiveness of the conjunctiva and eyelids is slightly greater than that of the surrounding skin of the face.

In most of the experiments the dose of roentgen rays was considerably greater than the so-called erythema or tolerance dose as used in the treatment of human beings. In some experiments the dose ranged from six to twenty-six times the erythema dose for human beings. Eyes exposed to smaller doses were not at all affected.

Cornea. — From many experiments the cornea appears definitely more resistant to irradiation than the conjunctiva and the lids. This conclusion is supported by the fact that the keratitis ap-

peared later, was usually less marked, and subsided sooner than the conjunctivitis. The effect of the rays on the cornea usually consisted in mere dulness, and this disappeared in a few days. Only after strong single doses or when the eyes were irradiated daily for some time was destruction of the cornea observed. The keratitis was characterized by irregular degeneration, destruction, and thinning of the corneal epithelium, which was reduced to one or two layers of cells with indistinct outlines, vacuolization of the protoplasm, and deformation or disintegration of the nuclei. Leukocytic infiltration, especially around the vessels in the vicinity of the limbus, and swelling of the endothelial lining of the vessels, were observed only after strong irradiation.

Iris. — The iris is even less sensitive to irradiation with roentgen rays than the cornea; in the rapidly growing eyes of young animals the only change observed after exposure was retardation in the formation of pigment; that is to say, the color changes in the iris took place more slowly. In some animals hyperemia, iritis with fibrinous exudate, loosening of the posterior pigment epithelium, and adhesions to the capsule of the lens were sometimes noted in eyes which had been exposed to exceptionally strong doses and in which severe conjunctivitis and keratitis had resulted. It is probable, therefore, that inflammation of the iris after irradiation is secondary to the conjunctivitis and keratitis and does not represent a specific reaction to the rays.

Crystalline lens. — If the radiosensitiveness of the conjunctiva, eyelids, cornea and iris is only slightly greater in young than in adult animals, the susceptibility of the lens is much greater. The experimental evidence shows that irradiation of the eye of young animals, soon after birth, may readily cause degenerative changes leading to cataract. The changes consist in granular and vacuolar degeneration of the lenticular epithelium, first around the periphery and later extending toward the center of the lens. This is accompanied by irregular desquamation of the anterior epithelium, and the patches of desquamated epithelium are separated by areas of cellular proliferation. The capsule slowly thickens, more or less atrophy of the lens supervenes, and the depth of the anterior chamber increases.

When the eyes of mature animals were irradiated, abnormalities of the lens were not observed in any of the early experiments. The results indicated that the mature lens is remarkably insensitive, even to doses of roentgen rays several times greater than the

doses ordinarily used in treating human beings. More recent investigations by Aulamo, Goldman, Rohrschneider,[133,134,135,136] Politzer, and Peter[119] have shown that the inference of great tolerance on the part of the mature lens is only partly confirmed. Prolonged observation and examination of the lens of irradiated animals by means of the slit-lamp have established the fact that the fully developed lens is somewhat less sensitive to irradiation than the conjunctiva and eyelids, and that a dose of roentgen rays below the limit of tolerance of the conjunctiva is not likely to affect the integrity of the lens.

As a result of his experimental observations, Rohrschneider justly stressed the point that perceptible changes in the lens were never found during the first three months following irradiation with roentgen rays but, after this latent period, opacification of the lens occurred in rabbits whose eyes had received a dose of 890 R* (100 per cent of the rabbit epilation dose, or 180 per cent of the human skin unit dose) or greater of rays filtered through 0.5 mm. of copper and 3 mm. of aluminium, or 980 R of rays filtered through only 3 mm. of aluminium. Cataract never resulted when smaller doses were employed. The latent period and the degree of lenticular opacity varied slightly from animal to animal, but was generally a function of the quantity and quality of the rays. Peter[119] also found lenticular opacification occurring from forty days to seven months after irradiation. On the strength of these observations, the conclusion was drawn that the crystalline lens is a highly radiosensitive structure. It is true that it is more sensitive than earlier experiments had led us to believe, but critical analysis of all the experimental evidence hardly warrants the excessive fears of Rohrschneider and others. The weight of evidence indicates that the lens of adult animals is slightly less radiosensitive than the conjunctiva and eyelids and that doses of roentgen rays within the tolerance of the conjunctiva and lids are not likely to induce deleterious effects in the lens.

Aqueous and vitreous humors. — The only changes observed have consisted in flakes of exudate and leukocytic infiltration, probably secondary to inflammatory reaction of other structures.

Retina. — As shown by the experiments of Tribondeau and Récamier, Belley, and Tribondeau and Belley,[153,154] the outstanding effect on the retina of young animals exposed to roentgen rays soon after birth is retardation of development affecting only the

* German R; not the international roentgen (r) now in use.

internal granular layer. Such retardation, together with retarded development of the eyeball as a whole, causes the external granular layer and the layer of rods and cones to assume a plicated appearance which is characterized by multiple folds of the anterior half or two-thirds of the retina. Sometimes these folds are so marked that the affected part of the retina appears to have an acinous formation. Moreover, the degree of plication corresponds roughly to the duration and intensity of irradiation and to the subsequent period of observation. No other alteration of the retinal coats was found.

Exposure of the fully developed eye of adult animals, even to doses of roentgen rays sufficient to cause marked conjunctivitis, dermatitis of the lids and keratitis, does not appear to induce any perceptible change in the retina. Birch-Hirschfeld[11] and Jacoby[22] have described what they regarded as pathologic alterations in the retina, which they ascribed to irradiation. These consisted in degeneration of some of the ganglion cells, with shrivelling and granular disintegration or actual disappearance of the nuclear chromatin, vacuolization of the protoplasm, loosening of the nerve fiber and inner plexiform layers, reduction in size of the inner granules, granular degeneration of the outer segment of the rods and cones, and myelin degeneration of the optic nerve. But other investigators, who gave to their animals doses even larger than those used by Birch-Hirschfeld and by Jacoby, did not observe such changes. Tribondeau and Lafargue[156] seem to have been justified in contending that the retinal alterations described by Birch-Hirschfeld and by Jacoby were probably due to improper histologic technic or were secondary to inflammation induced by irradiation in other more sensitive structures. The retina and optic nerve of animals more than two or three months old appear to possess remarkable resistance to roentgen rays.

Radium. — Many of the experiments undertaken to elucidate the action of radium on the eye have little value because the reports failed to specify essential technical details. It is therefore possible to draw only broad conclusions. In general the experimental evidence shows that the effect of radium irradiation on the ocular structures is substantially the same as that of roentgen rays.

CLINICAL RADIOTHERAPY (EYE)

Inflammatory conditions of eyelids. — Blepharitis. Evidence that simple blepharitis may be influenced favorably by suitable

doses of roentgen rays was furnished by Bunting, who reported the case of a patient with bilateral blepharitis complicated by recurring attacks of conjunctivitis and keratitis, who was completely cured by roentgen irradiation after having been treated unsuccessfully by various methods for two years. Marqués effected the cure of lupus vulgaris of the lids of a man aged eighty, and Hensen and Schäfer recorded the cure of three patients with chronic blepharitis associated with tuberculous conjunctivitis. Other examples of cure or improvement following exposure to roentgen rays have been given by Kumer and Sallmann, Lane, Pordes, and others. Kempster testified to the value of irradiation in trichiasis, and New and Benedict in blastomycosis of the lids (radium).

Follicular and Parinaud conjunctivitis. Among those who have observed the favorable action of roentgen rays or radium on these conditions may be mentioned Pardo, Jacoby,[73] Cohn, Uhthoff, Coutard and Offret, and Van Duyse. In some cases, no doubt, the favorable effect may have been only temporary; the evidence on this point is not conclusive.

Trachoma. Mayou[105] reported the case of a girl aged fourteen, with bilateral trachoma, whose left eye was cured by repeated exposure to roentgen rays. The right eye, treated with a solution of copper sulphate, improved much less, but roentgen irradiation completed the cure of this eye also. Later, Mayou[106] recorded nine other cases, all of the patients having been benefited by roentgen treatment, and five having been cured. In two cases Stephenson and Walsh exposed only one eye to roentgen rays. In both cases the disease disappeared from the irradiated eye, but that in the unirradiated eye continued unabated. Since then many other examples of improvement or cure of trachomatous conjunctivitis have been reported. In some cases, perhaps because of insufficient treatment, the lesions recurred; at any rate, resumption of treatment often resulted in cure. In many other cases recurrence did not take place.

Harman reported failure to influence trachoma with roentgen rays. Terrien admitted the regression of lymphoid hyperplasia and the disappearance of granulations, but complained that recurrence was the rule. It is possible that faulty technic may have accounted for the unsuccessful experience of these two authors. Derby also wrote disparagingly of the value of roentgen therapy; however, his opinion was not based on personal experience but on a cursory review of the literature. It is impossible to read the numerous communications on this subject, notably those of Cochard and of

Meldolesi and Sabbadini, without being convinced that roentgen rays have a real effect on the lesions of trachoma. The effect is greatest in the early, infiltrative stage of the granular form of the disease, and least when the granulations have been replaced by connective tissue.

Radium also has been found effective against the lesions of trachoma. To treat such a disease with a small quantity of radium is difficult; certainly irradiation with roentgen rays is a more simple procedure and less inconvenient or uncomfortable for the patient; with the latter agent, the exposure time is a matter of minutes, whereas, with the former, it is a matter of hours.

Tuberculous conjunctivitis. In this condition also, exposure to roentgen rays may be followed by substantial improvement but, as in tuberculous lesions in general, the effect of irradiation is characteristically slow, and repeated treatment at regular intervals is necessary to bring about maximum improvement.

Vernal conjunctivitis. Since the reports by Allport, Pusey, Pardo, Zentmayer, Gonzales,[53,54] and many others, many ophthalmologists regard radiotherapy as an effective method of dealing with this condition. In many of the cases reported the patients were cured. From the character of the disease it seems most likely that the chief factor in the action of roentgen rays or radium is the destruction of leukocytes infiltrating the conjunctiva. More patients have been treated with radium than with roentgen rays because a large proportion of them have been treated by ophthalmologists and not by radiologists. The factors of economy of time, relative discomfort and convenience to the patient undoubtedly favor roentgen irradiation. There is no sound basis for the idea that the action of radium is more rapid; this depends on the dose and on the technical conditions of irradiation.

Cornea. — Simple inflammation of the cornea appears to yield to roentgen rays much as does inflammation elsewhere, and the rays probably act in the same way. The favorable reports of Coleman, Sgrosso, and Lawson and Davidson seem quite convincing. According to F. H. and C. H. Williams, Linnell, Fellows and Lane, opacity of the cornea, when the inflammatory deposits have not been replaced by connective tissue, may be influenced favorably by exposure to roentgen rays or radium.

Fairly convincing evidence of the value of radiotherapy in corneal ulceration has been presented by Coover, Flemming, Lawrence, Lawson and Davidson, and others.

Judging by the reports of Japiot and Bussy,[75,76] Ramié, Guarini, Bywater and Plummer, Norton, Koster, and others, interstitial keratitis also appears amenable to roentgen rays or radium. Janeway, on the contrary, failed to observe improvement in one case.

As in tuberculous lesions in other structures and tissues, tuberculosis of the cornea has been shown to respond favorably to irradiation. Examples are to be found in the reports of Bettremieux, Mylius, Koster and Cath, and others. In some cases the lesions also affected the iris. Pardo, Coleman, Thiébault, and Wassing recorded the favorable action of roentgen rays or radium. In the case of one patient treated by the last author, tuberculous scleritis, keratitis and iridocyclitis disappeared completely after repeated irradiation.

Iris. — Coover, Sgrosso, Handmann, Pordes, and others, have shown that simple inflammation of the iris often yields to moderate doses of roentgen rays or radium. Moreover, in acute cases, a single exposure is sufficient to stop the inflammatory process.

Tuberculous iritis apparently responds as well to roentgen or radium treatment as similar inflammation of the conjunctiva and cornea. In the cases of Rollet and Bussy,[138,139] Jendralski, Birch-Hirschfeld,[14], Scheerer, Wetterstrand, and Martenstein and Richter, the results of treatment were most favorable when irradiation was started during the early, proliferative (infiltrative) phase of the lesions. From a fourth to a half of an erythema dose of roentgen rays generated at moderate voltage was repeated at intervals of two or three weeks. In the case of none of the twenty-nine patients dismissed as cured by Martenstein and Richter, did the disease recur, although some patients were followed for as long as eight years.

Lens and choroid. — In 1905 Norton attempted to treat immature cataract with radium, but failed. Later, Cohen and Levin, and Franklin and Cordes claimed to have treated successfully many patients with cataract, but the data furnished by them were not impressive. Moreover, a sound or even plausible basis for radiotherapy of cataract has never been advanced.

Darier, Koster, and Hernnheiser and Braun[66,67] have reported relief from pain soon after radium treatment of a patient suffering from choroiditis and periorbital neuralgia of long standing, improvement in vision after roentgen treatment of a patient with disseminate choroiditis, and complete cure of three patients with tuberculosis of the choroid after exposing the affected eye to one-

sixth of an erythema dose of roentgen rays of medium wave length (120 kilovolts, filtered through 4 mm. of aluminium) several times at weekly intervals.

Traumatic injuries of the eye. It would also appear that, in some cases at least, the inflammatory reaction following traumatic injuries of the eye is influenced by roentgen or radium irradiation. Among those who have observed favorable effects may be mentioned Axenfeld,[5] Viganò,[162] and Handmann. In six cases Viganò found irradiation useful in favoring the resolution of adherent scars and the absorption of hemorrhagic effusions, exudates and infiltrates.

Retina. — Circumscribed collections of extravasated blood (hematoma) and hemorrhagic effusions into hollow organs of limited size sometimes are influenced favorably by irradiation. Hessberg, with roentgen rays, successfully treated five patients with retinal hemorrhage, from trauma in one case, from arteriosclerosis in two cases, and from tuberculosis and syphilis, respectively, in the two remaining cases. Only one eye was irradiated, and the blood was said to have been absorbed more rapidly and vision to have improved more in the exposed than in the unexposed eye. Darier also noted rapid improvement after radium treatment in one case of hemorrhage into the vitreous, but Norton and Koster found radium treatment ineffective.

Glaucoma. It is not easy to understand how exposure of the eye to roentgen or radium rays could have any great or lasting effect on glaucoma. Nevertheless, Linnell, Guarini, Corbett, Hensen and Schäfer, Lloyd, and Deutsch reported rapid relief from pain and varying degrees of improvement. In some cases glaucoma was associated with hemorrhage, and the improvement may have resulted from the rapid absorption of exuded blood following irradiation. In other cases glaucoma may have been secondary to an inflammatory process, and improvement may have followed action of the rays on leukocytes infiltrating the inflamed area.

Injury to the eye from excessive irradiation. — Ocular injury from excessive irradiation has been recorded from time to time, but the number of such cases is surprisingly small. Most of the instances of radiation injury have been incidental to the treatment of ocular or orbital tumors with roentgen rays or radium and such injuries have been due to overexposure. In some of the cases reported by Cautley and Harman, and by Birch-Hirschfeld,[11] the apparent injury probably represented changes unrelated to irradi-

ation. However, in other cases described by Birch-Hirschfeld,[12,13] Ehnhuus, Fischoeder, Peter,[118] Flaschenträger, Rohrschneider,[137] and Knapp,[83] the relation of the injury to irradiation cannot be doubted. Irritative reaction took the form of epilation of eyelashes, conjunctivitis, keratitis, sometimes to the point of ulceration, iritis, and endothelial degeneration of the ocular blood vessels.

Small doses of roentgen rays repeated frequently for a long time may induce deleterious changes in the eye, but only two cases have been recorded, one by Sherer and one by Bennett. In the first case epilation of lids and brows, conjunctivitis and slight retinitis developed in the eyes of a physician who had been working with roentgen rays daily for three years. From the circumstances, however, it is likely that the disturbances noted were due, not to the cumulative effect of frequent exposure for a long time, but to a single, recent over-exposure. Under modern conditions, with our present knowledge of protection against radiation, injuries of this kind are unheard of.

Cataract. The experimental evidence indicates that the mature lens is less radiosensitive than the conjunctiva. The difference, however, is not great, and cataract is likely to develop in eyes exposed to excessive doses of roentgen rays or radium. This is especially true when full doses are concentrated on the eye through several fields, as in the treatment of intraocular or orbital tumors. In young children, the danger is even greater because the rapid growth of the lens during early life makes this structure so sensitive to irradiation that cataract may be induced even by moderate doses. Nevertheless, if a neoplastic process can thus be cured, even at the cost of cataract, the price cannot be regarded as exorbitant. But to induce cataract by radiotherapy for an inflammatory condition would be a different matter. Fortunately, the dose of rays most effective against acute or chronic inflammation is well below the limit of tolerance of the conjunctiva and lens.

Examples of injury from excessive irradiation have been reported by Gutmann, Treutler, Paton, Axenfeld,[4,6] Wilkinson, Pfahler,[120] Rohrschneider,[132,137] Knapp,[82,83] Kress, Robinson, and many others. In some cases the relation between cataract and irradiation is not at all clear, but in the majority the relationship can hardly be doubted.

Lacrimal apparatus. — Few experiments to test the radiosensitiveness of the lacrimal gland have ever been undertaken. Clinical data indicate that like other secretory glands, this acinous structure

is influenced in much the same manner by irradiation. Its specific sensitiveness, however, is less than that of some other glands, such as the salivary glands. The successful treatment of trachoma with obstruction of the duct, of chronic dacryocystitis, and of tuberculous dacryocystitis, has been reported by Falta, Viganò,[161] and Hensen and Schäfer.

Technic. — Acute inflammatory lesions. In acute inflammations, whether localized or diffuse, a single exposure to a small dose of roentgen rays is generally sufficient. By a small dose is meant a dose of from 10 to 50 per cent of the so-called erythema, or tolerance, dose for the skin of a young, adult, blond individual. In terms of the international quantitative unit of roentgen rays, such a dose would be specified as from 50 to 300 roentgens measured in air. In most cases roentgen rays generated at a potential between 125 and 150 kilovolts, and filtered through 4 mm. or 6 mm. of aluminium, are preferable to rays generated at 200 kilovolts and filtered through 0.5 mm. or more of copper or zinc. This is not intended to imply that rays of comparatively short wavelength generated under the latter conditions are devoid of therapeutic value. But it does mean that, in most cases, greater benefit can be obtained in less time with rays of comparatively long wavelength. The main reason for this advantage is that a larger proportion of rays of such quality are absorbed in the relatively superficial structures of the eye than in the case of rays of shorter wavelength. Necessarily, also, treatment with rays of medium wavelength requires less time and involves less inconvenience and discomfort for the patient. In some cases failure of the lesions to involute promptly may make it advisable to repeat the treatment after an interval of six or seven days.*

Comparable results may be obtained with corresponding doses of radium which may be used especially for patients who must be kept in bed and who cannot or should not be moved. The use of radium, however, has certain disadvantages. Inasmuch as the wavelength of the gamma rays is extremely short, and only a small percentage of such rays are absorbed in a given time by the tissues of the eye, the time of exposure must be considerably increased. Moreover, unless a considerable quantity of radium is available, uniform exposure of all parts of the eye (if this should be indicated) is more difficult than with roentgen rays.

* According to the experiments of Lacassagne this appears to be the optimum interval.

Chronic inflammatory lesions. The treatment of such conditions requires repeated irradiation with roentgen rays at intervals determined by the dose given at each exposure. This may mean two or three exposures, as in vernal conjunctivitis, or many doses over a period of time, as in tuberculous lesions. In chronic inflammatory processes, also, the dose of roentgen rays given at each session of treatment should be larger than for acute lesions, but should never be so large as to jeopardize, even temporarily, the integrity of the most sensitive of the ocular structures, the conjunctiva and the crystalline lens. According to the precise character of the lesion, the dose of rays may vary between 33 and 75 per cent of the erythema, or tolerance, dose (185 to 420 roentgens). Such doses may safely be repeated at intervals of from fifteen to thirty days. In tuberculous lesions, when individual doses of 420 roentgens are given at each session, the interval between sessions of treatment may be extended further. Like similar processes elsewhere, tuberculous processes affecting the eye respond favorably but slowly to irradiation, and treatment may have to be continued for some time.

Action of roentgen rays and radium. The weight of experimental and clinical evidence indicates that the first and major effect of the rays is destruction of some of the infiltrating leukocytes, especially the lymphocytes and polymorphonuclear cells which have long been known as the most radiosensitive of all cells. Moreover, the destruction of infiltrating leukocytes which occurs, or at least begins, soon after irradiation, probably serves to liberate the antibodies, ferments and other protective substances contained by these cells and to make these substances more available for defense than they were in the intact cells. Destruction of infiltrating leukocytes, also, tends to increase phagocytosis.

In chronic inflammatory lesions, other elements enter into the action of rays. Chief among these is the relative proportion of connective tissue organization (repair) and of degenerative changes, such as caseation and calcification. These factors probably account for the comparatively slow effect of irradiation.

Benign tumors of the eyelids. — Among the benign tumors of the eyelids the most common are angioma, nevus, verruca, molluscum and xanthelasma. Angioma and nevus of the lids behave like similar lesions elsewhere, and their response to radiotherapy is the same. Small cavernous angiomas can usually be cured by judicious irradiation, while large and deep-seated angiomas may

improve to varying degrees. Since this variety of lesion is most common in infants and children, it is essential that treatment should be planned in such a way as to avoid injuring the eyeball, and especially the lens. For small lesions roentgen rays of relatively long wavelength or, better still, radium (especially the beta rays) may be used. During roentgen or radium irradiation of an area of the lids the eyeball should be carefully protected. Effective protection can be provided by inserting beneath the lids, after preliminary anesthesia of the conjunctiva with a 1 per cent sterile solution of holocaine, carefully sterilized shields of lead shaped to conform to the curvature of the eyeball, and with smooth surfaces and edges.

The port-wine variety of nevus is not benefited by radiotherapy, but the strawberry variety is quite amenable to such treatment. In general, the beta rays of radium yield the best results, partly because such rays are absorbed by the tissues of the affected lid and partly because the special applicators used for such treatment are simple and convenient. The earlier in life such lesions are treated, the better the results.

Warts of the eyelids should be treated in the same way as warts in other parts of the body. Depending on their size and thickness, one or two erythema doses (560 or 1120 roentgens) administered to the lesion through a hole in a lead shield (with the eyeball separately shielded after preliminary anesthesia) is usually sufficient. Even if the wart or warts should disappear after the initial irradiation, it is usually wise to give a second treatment one month or six weeks later, but on the second occasion the dose should be somewhat smaller.

As for molluscum, a few cases have been reported in which patients were cured or improved by roentgen treatment but other patients have failed to respond. The same may be said of xanthelasma.

Malignant tumors of the eyelids. — The most common malignant neoplasms affecting the lids are epitheliomas of the basal-cell or squamous-cell type. Against the former, thorough roentgen or radium irradiation is effective in a large percentage of cases. But if recurrence is to be prevented, the entire lesion must be exposed to a maximum dose of rays so as to destroy every malignant cell. Every part of the lesion and a narrow zone of apparently normal tissue around it should be included in the field of irradiation, but the remainder of the lids as well as the eyeball itself should be carefully and adequately shielded from the rays.

As far as dosage is concerned, some prefer a massive single dose of unfiltered roentgen rays at one sitting, some a massive dose of rays of medium wavelength filtered through 4 mm. or 6 mm. of aluminium, some a strong dose of rays of short wavelength (200 peak kilovolts, with a filter of 0.5 mm. of copper or zinc); others continue to amuse themselves but finally disappoint the patient by treating such lesions with small, "safe" doses at varying intervals. At the present time the majority of experienced radiologists favor fractional irradiation, but opinions on the distribution of the treatment in relation to the time factor still differ considerably. The essential requirement is that the total dose must be sufficient to destroy all the malignant cells.

Without doubt radium can be used just as effectively, but radium therapy requires more time and is less comfortable for the patient.

In some cases squamous-cell epithelioma of the lids also can be treated successfully with roentgen rays or radium, but he who undertakes such treatment must be skillful and well trained, because in venturing to treat lesions of this kind he necessarily assumes a heavy responsibility. In general, the principle outlined in connection with the treatment of basal-cell epithelioma should be applied to irradiation of the primary lesion but, in addition, the lymphatic drainage of the region should also be irradiated. When the lesion is operable and when a skillful surgeon is available, however, it is perhaps wiser to advise thorough surgical removal. The circumstances in each case must govern the decision. If the lesion is dealt with by surgical means, the operative procedure should preferably be followed, as promptly as may be compatible with the condition of the patient, by thorough irradiation with roentgen rays. Such exposure must include every portion of the operative field and as much as possible of the lymphatic drainage (first group of lymph nodes). The fear entertained by some surgeons that postoperative irradiation interferes with healing of the wound is based on the degenerative tissue changes which sometimes occur after excessive doses of roentgen rays or radium. However, if the radiotherapeutic procedures are closely supervised by an experienced radiologist, the healing process should be neither prevented nor even retarded.

Benign tumors of the conjunctiva and cornea. — What has been written concerning angioma and nevus of the lids applies to similar lesions of the conjunctiva, but the treatment of such lesions

with roentgen rays or radium requires even greater care in protecting the adjacent normal tissues. As far as radium treatment is concerned, applicators specially prepared to meet conditions peculiar to each case may be required.

As for desmoid or lipoma of the conjunctiva, such growths are not amenable to radiotherapy and should be removed surgically. The same is true of cysts, although some cysts are lined with cells that may be moderately radiosensitive. Polyps also should preferably be treated by surgical methods, but according to their histologic character some lesions of this kind are influenced more or less by exposure to roentgen rays or radium. The cellular hyperplasia characterizing papilloma of the conjunctiva, especially during its early stages, accounts for the radiosensitiveness of a certain proportion of such growths. The decision between surgical treatment and radiotherapy must be governed by the circumstances in each case. Fibroma and myxoma of the conjunctiva are just as resistant to irradiation as similar lesions in other parts of the body. Lymphangioma, on the contrary, may be successfully treated by exposure to roentgen rays or radium.

Malignant tumors of the conjunctiva and cornea. — Malignant tumors of this structure usually are epitheliomas or sarcomas. Epithelioma often arises from an antecedent papilloma; it is usually situated in the region of the limbus, although it may extend into the cornea. As extension proceeds, the growth penetrates the eyeball along the course of the vessels and may finally destroy the sclera. Only during the early phases of the process may such lesions be successfully treated with roentgen rays or radium. Radium is distinctly preferable because, by means of specially constructed applicators, a dose sufficient to destroy the neoplastic cells may be given without injury to the adjacent ocular tissues.

Like similar tumors elsewhere, melano-epithelioma of the conjunctiva is quite resistant to irradiation, and the prognosis in such cases is decidedly bad.

Primary tumors of the cornea are rare. Usually neoplasms that invade the cornea originate in the adjacent conjunctiva.

Tumors of the iris. — The newgrowths which are known to occur in the iris are cysts, melano-epithelial growths, and sarcomas. Cysts should preferably be excised. Early radical removal is the therapeutic method of choice for melanotic and sarcomatous tumors, but the results cannot be said to warrant enthusiasm.

Tumors of the choroid. — These neoplasms usually are melanoepitheliomas, often miscalled sarcomas. As in melanotic tumors generally, radiotherapy has comparatively little effect on such growths, which are highly malignant and resist irradiation.

Tumors of the retina. — Glioma is the usual form taken by newgrowths of this structure, and the gliomatous process tends to destroy the eye and may extend along the optic nerve backward to the brain. It occurs only in children, and the prognosis is decidedly unfavorable. If the neoplasm is confined to the eyeball, prompt removal may effect a cure. Roentgen or radium irradiation may check the growth of the tumor, relieve pain, and may even induce a considerable degree of regression, but few patients can thus be cured. Recently, Martin and Reese have shown that skillful roentgen treatment may cause the malignant process to disappear, and some of their patients have remained well.

EXPERIMENTAL BACKGROUND (EAR)

Only a few experiments to test the sensitiveness of the auditory apparatus to roentgen rays have been conducted, and the experiments with radium have been confined to its effects on the labyrinth. The early investigations of Thielemann and Ewald did not throw much light on the subject, because their failure to give detailed information robbed their work of value. On close analysis, the conclusions which Marx drew from his experiments are not sustained by his own data. The abnormalities observed and described by Marx were not related to radium irradiation, but probably were due to the mechanical effect of the radium container or to the operative intervention required to insert the radium into the labyrinth. The experiments of Chilow also were inconclusive. It is apparent, therefore, that the experimental evidence is quite meager. Certainly, there is nothing to indicate that the normal auditory apparatus is sensitive to roentgen or radium rays.

CLINICAL RADIOTHERAPY (EAR)

Inflammatory processes. — Eczema of the outer part of the ear responds to irradiation much as does eczema in other parts of the body. Acute eczema usually subsides promptly after a moderate dose of roentgen rays. Chronic eczema, on the other hand, may require several exposures to small or moderate doses at intervals governed by the dose. Acute perichondritis, like many other acute inflammatory processes, often improves rapidly after appropriate

irradiation. The more acute the lesion, the smaller need be the dose of roentgen rays or radium.

Thost and Urbantschitch have been the only ones who have written about radiotherapy for this condition. The first recorded the cure of two patients with roentgen rays, and the latter of one patient (tuberculous ulcers of the concha) with radium.

Although the fact is little known, hematoma of the ear, so common among pugilists but occurring from any sharp blow on the ear, often responds quite well to roentgen or radium treatment. Here also moderate doses are in order. Forschner and Regnier have reported successful results in twenty-three cases. Few of their patients had to be treated more than once.

Evidence that roentgen irradiation may be effective against chronic inflammation of the middle ear is fairly abundant. In this connection the reports of Dionisio (twenty cases), Beattie (twenty-nine cases), Raynal (three cases), Yocom (thirty-five cases), and Di Donato (seventy-five cases) are interesting. These reports indicate that a certain percentage of patients may derive benefit from, and that some may be cured by, appropriate irradiation. Moderate doses, repeated at intervals depending on the dose of rays, would appear to be most effective.

Granger and others have shown that, when exposed to a small dose of roentgen rays early in the course of the disease, acute mastoiditis often resolves spontaneously without suppuration, and the serious operation of radical mastoidectomy may thus be avoided.

It is difficult to understand how a disease such as otosclerosis can be influenced to any great extent by roentgen or radium rays. Yet a number of reports of favorable results from irradiation have been published by Jaulin, Schwarz, Webster, Lüdin, Grande, Kriser, Révész and Kövari, Lake, Bryant, and others. The evidence furnished is not impressive, and some of the reports are obviously worthless. Nevertheless, a certain proportion of patients appear to have been unquestionably benefited. It is probable that the cases in which radiotherapy is followed by improvement are those in which the disease is associated with chronic inflammatory changes and a measure of leukocytic infiltration. This would explain the improvement.

Many also have claimed improvement in hearing and relief from tinnitus after exposure of the ear to roentgen rays or radium. Among these may be mentioned McCulloch, Bryant, Boonacker, Jarvis, Kinney, Albrecht, Meyjes, Haenlein, Burger, and others. These reports are difficult to evaluate, however, because so few

contained adequate data. If the improvement noted was indeed related to irradiation, it seems likely that this occurred in cases in which the auditory disturbances were probably caused by lymphoid hyperplasia in the throat or by inflammatory processes in the ear, throat and nasopharynx, with associated leukocytic infiltration.

If the reports of Cemach and of Bondy can be accepted as representative, tuberculosis of the middle ear appears to respond as favorably to roentgen irradiation as tuberculous processes affecting many other structures of the body. Moderate doses of roentgen rays repeated at regular intervals should cause the gradual involution of such lesions.

Benign tumors. — Sebaceous cysts and fibromas of the auricle are not amenable to radiotherapy and should preferably be treated by surgical methods. Angioma cavernosum may be treated successfully with roentgen rays or radium, but the treatment must be carefully planned and skillfully given so as to avoid injury to the auricular cartilage or breakdown of the overlying skin. Nevus of the strawberry type also can be treated successfully by roentgen, or preferably by radium, irradiation. A flat applicator of half-strength, with the radium filtered through 0.1 mm. of aluminium, should be held over the lesion, or paraded over a part of the lesion, if it is large, at a distance of 1 cm. from the skin for fifteen minutes. This may have to be repeated two or three times at intervals of one month.

Malignant tumors. — Epithelioma of the auricle or auditory canal is an uncommon tumor and usually arises on a background of chronic eczema, chronic purulent infection, or other variety of chronic inflammation. Early surgical removal is usually resorted to, and an occasional cure is thus obtained; in many cases, however, the tendency to infiltration and ulceration, with secondary infection, leads to early metastasis to the cervical nodes. In addition, such growths are prone to extend toward the middle ear, to invade and destroy the tissues around the canal, and the surrounding bones (petrous portion of the temporal bone) may thus be destroyed.

When seen early, thorough and skillful exposure to roentgen rays or radium may cause the neoplastic process to retrogress, and in some cases patients have thus been cured. In the majority of cases, however, a period of slight or marked improvement is followed by fresh malignant activity, with rapid extension. In irradiating the auricle, excessive doses must be avoided because of the

relative ease with which severe injury to the auricular cartilage can be produced.

Sarcoma seldom arises in the auricular structures themselves but usually extends to the ear from adjacent parts. Its reaction to irradiation depends on its histologic composition, and it behaves like corresponding sarcomas in other structures.

EXPERIMENTAL BACKGROUND (NOSE AND ACCESSORY SINUSES)

The radiosensitiveness of the mucous membrane of the nose and sinuses has not been tested experimentally, and what is known on the subject is based on clinical observation. From the standpoint of comparative sensitiveness to roentgen rays or radium, the mucous membrane of the nose and sinuses probably resembles that of the mouth and pharynx. However, since epithelial cells which have the property of secreting mucus have been shown to be more sensitive to irradiation than ordinary epithelium, and since a proportion of the cells lining the nose and sinuses secrete mucus, these cells undoubtedly react in much the same way to irradiation as do the mucous cells of the salivary glands, bronchi and small intestine. After exposure to certain doses of roentgen rays or radium, therefore, mucoid degeneration of the mucous secreting cells, with increase in the secretion for one or two weeks, is to be expected. Then the production of mucus falls below normal. The degree and duration of this effect depend on the dose of rays.

CLINICAL RADIOTHERAPY (NOSE AND ACCESSORY SINUSES)

Rhinitis and sinusitis. — Reports of the therapeutic value of roentgen rays and radium for certain pathologic conditions of the nose and accessory sinuses have been made. Osmond treated twelve patients suffering from acute or chronic inflammation of the antrum and frontal sinus and claimed that the inflammation subsided rapidly after small doses of roentgen rays. Pain was quickly relieved, the discharge gradually diminished and ceased, and the patients remained well. Denier treated with roentgen rays ten patients with frontal or maxillary sinusitis. In eight of the ten cases the headache, the secretion and mucous congestion disappeared promptly. In one case the headache did not disappear completely and in another the coryza recurred.

Robinson treated by combined surgical methods and radium fifty patients with polypoid sinusitis. In seven cases the asthmatic manifestations were relieved, the mucous or mucopurulent secre-

tion diminished, the tendency to headache abated, and a few patients recovered their sense of smell.

More recently, Butler and Woolley reported having treated 700 cases of chronic sinusitis, with marked improvement or apparent cure in a large proportion. Whether their experience will be confirmed by that of others remains to be seen.

Hay fever. — Only a few attempts have been made to test the possible value of roentgen irradiation for hay fever. Schmidt, who was a victim of hay fever, had given to himself one-third of an erythema dose of rays of medium wavelength over the nostrils. The sneezing disappeared in a few days, but recurred two weeks later; fresh irradiation had little effect. The symptoms of a patient who was irradiated twice were reported to have improved, but the duration of improvement was not specified. Park reported relief from symptoms for three years in the case of a patient who had been treated over a period of six weeks. Fresh symptoms then returned, and repetition of treatment was again followed by cessation of symptoms. The second patient, who had had annual attacks for thirty years, was treated over a period of four weeks and did not have any attack that year. Others, including Laquerrière, and Haag and Schreus, recorded favorable results in about half of the cases.

On the whole, the evidence is too meager to justify enthusiasm.

Ozena. — Much the same may be written concerning this condition. The reports of Yoel, Carrari, and Grauer are not impressive.

Rhinoscleroma. — On this form of chronic infectious granuloma, on the contrary, radiotherapy, at least during the hyperplastic and infiltrative stages, has been shown to exert a real influence. Judging by the reports of Fittig, Ranzi, von Schrötter, Freund, and others, the lesions of this disease appear singularly amenable to roentgen irradiation. As in other chronic inflammatory processes, however, the treatment must be repeated at intervals for some time.

Tuberculosis of the nasal mucous membrane. — As in other tuberculous processes, repeated exposure to a moderate dose of roentgen rays or radium has a favorable action. Havens reported five cases, in three of which the patients were treated with radium. Of these, two were well four and a half months and four years, respectively, following treatment.

Benign tumors. — The only benign tumors of the nose are polyps and angiomas, usually of the cavernous variety; the former are comparatively common, while the latter are decidedly uncommon. Polyps may be favorably influenced by radium or roentgen therapy, but only during the early stage, when a considerable element of inflammatory infiltration or lymphoid hyperplasia may be present. Later, when the polyps are made up chiefly of fibrous connective tissue, the effect of irradiation is slight or nil.

Malignant tumors. — Basal-cell and squamous-cell epitheliomas are the common neoplasms arising in the skin of the nose. The basal-cell variety of epithelioma may be cured in a large percentage of cases by thorough treatment with roentgen rays or radium. Of the squamous-cell variety a smaller percentage can thus be cured, because this kind of epithelioma is so prone to invade the lymph nodes draining the affected part of the nose. C. L. and J. H. Martin, Pfahler,[121] and others, have shown that skillful radiotherapy often succeeds in destroying the malignant process.

By thorough treatment is meant that the primary lesion, and the metastatic nodes, if any, must receive an adequate total dose of rays. Usually this can be accomplished by fractional irradiation. Whether the radiologist adopts the Regaud principle or some other scheme of fractional doses is of small consequence, provided a sufficient total dose of rays is delivered to the malignant cells within a given time. Unlike similar lesions in other parts of the body, the treatment of nasal epitheliomas is rendered even more difficult by reason of the nasal cartilage or nasal bones. Excessive or ill-planned irradiation may irreparably injure these structures, and the unfortunate condition of the patient may be made even worse. This is why the treatment of nasal epithelioma with roentgen rays or radium should be undertaken only by radiologists who have had special training and experience.

Of the malignant tumors that arise in the nasal mucous membrane, the squamous-cell epithelioma again is the most common. Some of these neoplasms can be cured by skillful surgery or with roentgen rays or radium. In a large proportion of cases, unfortunately, the malignant process recurs or an excellent immediate result is spoiled by metastasis.

In the nasopharynx, the squamous variety of epithelioma is perhaps the most common, although lymphoid, lympho-epitheliomatous, or transitional cell tumors also are common. At the present time divergence of opinion among pathologists concerning the dis-

tinctive features is still prevalent. Certain tumors arising primarily in the nasopharynx, and regarded by many pathologists as essentially epithelial in character, metastasize like lymphoid tumors and respond to irradiation like lymphoid and not like epithelial newgrowths. Obviously, extended study will be required to clarify this situation.

The lymphoid, lympho-epithelial and transitional cell tumors of the nasopharynx are much more radiosensitive than the unalloyed squamous epithelioma. Regardless of the variety, however, a fair percentage of such neoplasms can be cured, and in a large percentage of cases marked improvement can be obtained by thorough treatment with roentgen rays or radium, provided metastasis has not yet occurred. Even in the presence of metastasis, if this is not extensive, pronounced amelioration may follow the treatment. It is in connection with epithelioma in this situation, as also in the pharynx and larynx, that the principle of fractional irradiation, as developed by Regaud and applied by Coutard, has thus far yielded its best results.

REFERENCES

1. Albrecht, W.: *Ueber Radiumbehandlung bei Schwerhörigen.* Beitr. z. Anat. Physiol., Path. u. Therap. d. Ohres. 7:413-422, 1913-1914.
2. **Allport, Frank**: *A case of vernal conjunctivitis.* Ophth. Rec. 12:484-487, (Oct.) 1903.
3. Aulamo, R.: *Experimentelle Untersuchungen über die Wirkung der Röntgenstrahlen auf das Kaninchenauge.* Acta ophth. 5-6:489-490, 1927-1928.
4. Axenfeld, Th.: *Intraokuläre Strahlentherapie besonders beim Glioma retinæ.* Ber. u. d. Versamml. d. Ophth. Gesellsch. 396-409, 1916.
5. Axenfeld, Th.: *Kombinierte Operative und Strahlentherapie traumatischer sog. Iriszysten.* Deutsch. med. Wchnschr. 43:1182-1183, (Sept. 30) 1917.
6. Axenfeld, Th.: *Weitere Erfahrungen über intraokuläre Strahlentherapie.* Ztschr. f. Augenheilk. 40:109-111, 1918.
7. Beattie, Robert *Treatment of sub-acute and chronic otitis media with the use of the x-ray.* Jour. Michigan State Med. Soc. 20:449-451, (Nov.) 1921.
8. Belley, G. H.: *Étude expérimentale de l'action des rayons X sur l'œil en voie de développement.* Bordeaux, 1907, 86 pp.
9. Bennett, H. C.: *Roentgen-ray conjunctivitis.* Jour. Phys. Therap. 1: 384-386, 1905-1906.
10. Bettremieux: *Rayons X en thérapeutique oculaire.* Clin. Opht. 9:225-227, 1903.
11. Birch-Hirschfeld, A.: *Die Wirkung der Röntgen- und Radiumstrahlen auf das Auge.* Arch. f. Ophth. 59:229-310, 1904.

12. Birch-Hirschfeld, A.: *Weiterer Beitrag zur Wirkung der Röntgenstrahlen auf das menschliche Auge.* Arch. f. Ophth. 66:104-119, 1907.
13. Birch-Hirschfeld, A.: *Zur Wirkung der Röntgenstrahlen auf das menschliche Auge.* Klin. Monatsbl. f. Augenh. 46:129-137, 1908.
14. Birch-Hirschfeld, A.: *Die therapeutische Verwendung der strahlenden Energie in der Augenheilkunde.* Ztschr. f. Augenh. 49:48-50, 1922-1923.
15. Bondy, I. G.: *Geheilte Mittelohrtuberkulose.* Monatsch. f. Ohrenh. 62: 237, 1927.
16. Boonacker: Discussion. Nederl. Tijdschr. v. Geneesk. 58:2079-2083, 1914.
17. Bossuet, A.: *Experimentelle Untersuchungen über die Einwirkung der Röntgenstrahlen auf die Linse.* Arch. f. Augenh. 64:277-285, 1909.
18. Bryant, W. S.: *Notes on the use of radium in treatment of diseases of the ear.* Med. Rec. 86:376-377, (Aug. 29) 1914.
19. Bunting, T. L.: *Roentgen rays in the treatment of ulcerative blepharitis.* Lancet. 1:1014, (April 13) 1907.
20. Burger, H.: *Nadeelig gevolg van radium-behandeling bij hardhoorendheid.* Nederl. Tijdschr. v. Geneesk. 1:229-232, 1916.
21. Butler, F. E., and Woolley, I. M.: *Roentgen therapy in chronic sinusitis: a further report.* Radiology. 23:528-537, (Nov.) 1934.
22. Bywater, H. H., and Plummer, F. C.: *Preliminary note on the x-ray treatment of interstitial keratitis.* Brit. Med. Jour. 2:1152, (Dec.) 1923.
23. Carrari, G. G.: *Sulla radium-terapia dell ozena.* Valsalva. 3:77-82, (Feb.) 1927.
24. Cautley, Edmund, and Harman, N. B.: *Optic neuritis, (?) due to x-rays.* Proc. Roy. Soc. Med. Part 1, 9:3-5, 1916.
25. Cemach, A. I.: *Die Probleme der Röntgentherapie der Mittelohrtuberkulose.* Ztschr. f. Hals-, Nasen-, u. Ohrenh. 3:285-294, 1922.
26. Chalupecký, H.: *Ueber die Wirkung der Röntgen-strahlen auf das Auge und die Haut.* Centralbl. f. prakt. Augenh. 21:234; 267, 1897.
27. Chilow, K. L.: *Ueber die Anwendung von Radiumemanation in der experimentellen Labyrinthologie.* Monatschr. f. Ohrenh. 61:786-794, (July) 1927.
28. Cochard, M.: *La radiothérapie du trachome.* Lyon, Thèse, 1921, 59 pp.
29. Cohen, Martin, and Levin, Isaac: *The action of radium on cataracts.* Jour. Am. Med. Assn. 73:1193-1200, (Oct.) 1919.
30. Cohn, Hermann: *Die Heilung des Trachoms durch Radium.* Berl. klin. Wchnschr. 42:22, (Jan.) 1905.
31. Coleman, W. F.: *The x-ray and some personal observations of its use in diseases of the eye.* Jour. Ophth. and Oto-Laryngol. 1:125-133, 1907.
32. Coover, D. H.: *Roentgen ray flashes or intermittent x-rays in the treatment of eye diseases with report of cases.* Ophth. Rec. 18:331-338, (July) 1909.
33. Corbett, J. J.: *The effect of radium on glaucoma.* Boston Med. and Surg. Jour. 190:1124-1126, (June) 1924.
34. Coutard and Offret: *Traitement de la conjonctivite folliculaire par les rayons X très filtrés.* Ann. d'ocul. 157:240-244, 1920.
35. Darier, A.: *Röntgenstrahlen und Becquerelstrahlen in der Augentherapie.* Wchnschr. f. Therap. u. Hyg. d. Auges. 6:409, 1902-1903.
36. Denier: *Traitement des sinusites chroniques par la radiothérapie.* Bull. off. Soc. franç. d'électrothér. et de radiol. (June) 1929. Abst.: Jour. radiol. et d'électrol. 14:266, 1930.
37. Derby, G. S.: *X-ray and radium in the non-malignant diseases of the eye.* Boston Med. and Surg. Jour. 191:20-21, (July) 1924.
38. Deutsch: *Radiumbestrahlung bei hämorrhagischen Glaukom.* Klin. Monatskl. f. Augenh. 80:104, 1928.

39. Di Donato, D.: *Della roentgenterapia nelle otorree.* Arch. ital. d. otol. 37:335-337, 1926.
40. Dionisio, J.: *Traitement de l'otite moyenne suppurée chronique par les rayons X.* Arch. d'élect. méd. 14:934, 1906.
41. Ehnhuus, E.: *Et Tilfælde af overfladiske og dybe øjenlæsioner opstaaede under Bestraaling med. Røntgenstraaler.* Hosp.-Tid. 52:1228-1230, 1919.
42. Ewald: *Die Wirkung des Radiums auf das Labyrinth.* Zentralbl. f. Physiol. 19:297-298, (Aug.) 1905.
43. Falta, M.: *Das Radium in der Trachomtherapie.* Wien. med. Wchnschr. 55:1546-1551, 1905.
44. Fellows, C. G.: *Clinical reports on the roentgen ray flashes.* Homeop. Eye, Ear and Throat Jour. 16:389-391, 1910.
45. Fischoeder, Eduard: *Über Schädigungen am vordern Augenabschnitt durch Röntgenstrahlen an einem wegen Aderhautsarkoms bestrahlten Bulbus.* Ztschr. f. Augenheilk. 44:160-166, 1920.
46. Fitting, Otto: *Ueber einen mit Röntgenstrahlen behandelten Fall von Rhinosklerom.* Beitr. z. klin. Chir. 39:155-161, (June) 1903.
47. Flaschenträger, Heinrich: *Beitrag zur Radium- und Röntgenschädigung des menschlichen Auges.* Klin. Monatsbl. f. Augenh. 72:645-648, 1924.
48. Fleuring: *Experimentelle und klinische Studien über den Heilwert radioaktiver Strahlen bei Augenerkrankungen.* Arch. f. Ophth. 84: 345-400, 1913.
49. Forschner, L., and Regnier, E.: *Die Röntgentherapie des Othämatoms.* Strahlentherapie. 21:91-95, 1925-1926.
50. Franklin, W. S., and Cordes, F. C.: *Radium for Cataract.* Am. Jour. Ophth. 3:643-647, 1920.
51. Freund, L.: *Einen Fall von Rhinosklerom.* Wien. klin. Wchnschr. 18: 647-648, (June 15) 1905.
52. Goldman, Hans: *Über Entstehung von Diskontinuitätsflächen in der Linse.* Arch. f. Ophth. 122:198-218, 1929.
53. Gonzales, L. J. de J.: *Los rayos x en la terapeutica de la periqueratoconjunctivitis exuberante.* Rev. cubana de oftal. 2:125-131, 1920.
54. Gonzales, L. J. de J.: *Vegetación gigante reincidente de periqueratoconjunctivitis, que invadió la cornea y cedió a los rayos x.* Rev. cubana de oftal. 2:131-133, 1920.
55. Grande, C.: *La cura delle otiti medie catarrali coi raggi Roentgen.* Arch. ital. d. otl. 36:197-205, 1925.
56. Granger, Amédée: *The treatment of infections of the mastoids of infants, when there is no sign of destruction present, with fractional doses of the roentgen rays.* New Orleans Med. and Surg. Jour. 84: 111-113, (Aug.) 1931.
57. Grauer, S.: *Ein Beitrag zur Röntgenbehandlung der Ozæna.* Strahlentherapie. 31:801-805, 1929.
58. Guarini, Carlo: *La cura roentgen di alcune lesioni dell'occhio con le radiazioni a cortissima lunghezza d'onda.* Riforma med. 38:963-964, (Oct.) 1922.
59. Gutmann, G.: Discussion. Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. 32:337-338, 1905.
60. Haag, F. E., and Schreus, H.: *Über die Röntgenzehandlung des Heufiebers.* Strahlentherapie. 42:485-491, 1931.
61. Haenlein, Oskar: *Radium und Mesothorium bei Schwerhörigkeit und Ohrensausen.* Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres. 7: 423-449, 1913-1914.

62. Handmann, M.: *Vollständige Rückbildung einer traumatischen Iriszyste nach Röntgenbestrahlung.* Klin. Monatsbl. f. Augenh. 72:111-116, 1924.
63. Harman, N. B.: *Electrical treatment of trachoma.* Brit. Med. Jour. 2:435, 1905.
64. Havens, F. Z.: *Primary tuberculosis of the nasal mucous membrane.* Arch. Otolaryngol. 14:181-185, (Feb.) 1931.
65. Hensen, H., and Schäfer, Herm.: *Über die Ergebnisse der Röntgenstrahlenbehandlung bei Augenkrankheiten bzw. Tumoren des Sehapparates.* Arch. f. Ophth. 114:123-156, (Aug.) 1924.
66. Herrnheiser, G., and Braun, G.: *Die Röntgenbehandlung der Augentuberkulose.* Strahlentherapie. 28:770-778, 1928.
67. Herrnheiser, G., and Braun, G.: *Röntgentherapie der Augentuberkulose.* Fortschr. a. d. Geb. d. Röntgenstrahlen. 37:287, 1928.
68. Hessberg, R.: *Die Behandlung von Netzhautblutungen mit Röntgenstrahlen.* Strahlentherapie. 23:313-317, 1926.
69. v. Hippel, E.: *Ueber eine neue biologische Wirkung der Röntgenstrahlen.* Deutsch. med. Wchnschr. 31:2085-2086, (Dec.) 1905.
70. v. Hippel, E.: *Über angeborene Liddefekte bei neugeborenen Kaninchen nach Röntgenbestrahlung des trächtigen Muttertieres.* Ber. a. d. Ophth. Gesellsch. 33:293-295, 1906.
71. v. Hippel, E.: *Ueber experimentelle Erzeugung von angeborenem Star bei Kaninchen nebst Bemerkungen über gleichzeitig beobachteten Mikroophthalmus und Lidcolobom.* Arch. f. Ophth. 65:326-360, 1907.
72. Jacoby: *Experimentelle Untersuchungen über Schädigungen des Auges durch Röntgenstrahlen.* Strahlentherapie. 16:492-506, 1923-1924.
73. Jacoby, F.: *Zur Radiumbehandlung des Trachoms.* Deutsch. med. Wchnschr. 32:61-64, (Jan.) 1906.
74. Janeway, H. H.: *The therapeutic use of radium in diseases of the eye.* Arch. Ophth. 49:156-174; 238-243, 1920.
75. Japiot, P., and Bussy, L.: *La radiothérapie dans la kératite interstitielle.* Jour. radiol. et d'électrol. 5:106-112, 1921.
76. Japiot, P., and Bussy, L.: *La roentgenthérapie dans certaines affections inflammatoires de l'œil.* Paris méd. 47:126-128, (Feb. 3) 1923.
77. Jarvis, D. C.: *The effect of small doses of roentgen rays in certain forms of impaired hearing.* Am. Jour. Roentgenol. 10:201-202, (Sept.) 1923.
78. Jaulin: *Traitement de l'otite scléreuse par les rayons X.* Arch. d'élect. méd. 16:92-97, 1908.
79. Jendralski, F.: *Radiotherapeutische Erfahrungen bei Tumoren und Tuberkulose des Auges und seiner Umgebung.* Klin. Monatsbl. f. Augenh. 65:565-573, 1920.
80. Kempster, Christopher: *The treatment of trichiasis by means of the X rays.* Brit. Med. Jour. 1:332, (Feb.) 1915.
81. Kinney, L. C.: *The roentgen therapy of tinnitus aurium.* Am. Jour. Roentgenol. 10:378-379, (Sept.) 1923.
82. Knapp, Arnold: *Orbital changes produced by radium in cancer of the upper jaw.* Jour. Am. Med. Assn. 81:1849-1850, (Dec. 1) 1923.
83. Knapp, Arnold: *A series of radium injuries to an eye.* Arch. Ophth. 7:619-620, (Jan.) 1932.
84. Koster, W.: *Über die direkte Behandlung von Augenkrankungen mit Radium und Mesothorium.* Strahlentherapie. 3:582-598, 1913.
85. Koster, W., and Cath, I. G.: *Behandeling van oogziekten met radium.* Nederl. Tijdschr. v. Geneesk. 2:633; 702, 1911.

86. Kress, G. H.: Abstract of discussion. Jour. Am. Med. Assn. 81:1850, (Dec. 1) 1923.
87. Kriser: *Die Behandlung der Otosklerose mit Röntgenstrahlen.* Fortschr. a. d. Geb. d. Röntgenstrahlen (Kongressheft). 16:86-88, 1925.
88. Kumer, L., and Sallmann, L.: *Über die Radium-Therapie tuberkulöser Erkrankungen der Lider, der Bindehaut und des Tränensackes.* Ztschr. f. Augenheilk. 54:11-25, 1924.
89. Lacassagne, A., and Vinzent, R.: *Action des rayons X sur un foyer infectieux local, provoqué chez le lapin par l'injection de Streptobacillus caviæ.* Compt. rend. Soc. de biol. 100:247-249, (Jan. 26) 1929.
90. Lake, R.: *A case of early otosclerosis treated with radium.* Tr. Internat. Otol. Cong. 9:89-96, 1912.
91. Lane, Laura A.: *Radium in ophthalmology; with special reference to its use in benign affections.* Jour. Am. Med. Assn. 83:1838-1945, (Dec.) 1924.
92. Laquerrière, A.: *Rayons X et rhume des foins.* Bull. et mém. Soc. de radiol. méd. de Paris. 138:137, (April) 1927.
93. Lawrence, H.: *Radium in the treatment of eye cases.* Intercolonial Med. Jour. Australas. 14:566-567, 1909.
94. Lawson, Arnold, and Davidson, J. M.: *Radium therapy in eye diseases.* Brit. Med. Jour. 2:1491-1497, (Nov.) 1910.
95. Linnell, E. H.: *Roentgen ray flashes in the treatment of serious ocular affections.* Homeop. Eye, Ear and Throat Jour. 15:394-409, 1909.
96. Little, C. C., and Bagg, H. J.: *The occurrence of two heritable types of abnormality among the descendants of x-rayed mice.* Am. Jour. Roentgenol. 10:975-989, 1923.
97. Lloyd: *Le traitement de certains glaucomes par les rayons X.* Clin. Opht. 10:546, 1927.
98. Lüdin, M.: *Die Röntgentherapie in der inneren Medizin.* Schweiz. med. Wchnschr. 51:1081-1084, (Nov. 24) 1921.
99. Marqués, P.: *Lupus palpebral primitivo; radioterapia; curación.* Arch. de oftal. Hispano-Am. 8:333, 409-415, 1908.
100. Martenstein, H., and Richter, A.: *Erfahrungen über die Behandlung der Iristuberkulose mit Röntgenstrahlen.* Klin. Monatsbl. f. Augenh. 78:643-650, 1927.
101. Martin, C. L.: *Squamous cell carcinoma of the skin.* Am. Jour. Roentgenol. 28:728-737, (Dec.) 1932.
102. Martin, J. M.: *Fifteen years' experience with the fractional dose method of treating cutaneous malignancies.* Am. Jour. Roentgenol. 10:726-733, (Sept.) 1923.
103. Martin, H. E., and Reese, A. B.: *Treatment of retinal gliomas by the fractionated or divided dose principle of roentgen radiation: a preliminary report.* Arch. Ophth. 16:733-761, (Nov.) 1936.
104. Marx, Hermann: *Untersuchungen über experimentelle Schädigungen des Gehörorganes.* Ztschr. f. Ohrenh. 59:192-210, 1909.
105. Mayou, M. S.: *A case of trachoma treated by x-rays.* Tr. Ophth. Soc. U. Kingdom. 22:95-96, 1902.
106. Mayou, Stephen: *The treatment of trachoma by the x-rays.* Tr. Ophth. Soc. U. Kingdom. 23:10-23, 1903.
107. McCulloch, H. D.: *Extreme deafness (labyrinthine) showing improvement by treatment with the ultra X rays.* Lancet. 2:223-224, (July 26) 1913.
108. Meldolesi, G.: *Sulla terapia del tracoma con le radizioni secondarie (secondo il metodo del Ghilarducci).* Actinoterapia. 4:97-162, 1924.
109. Meyjes, W. P.: *Doofheid behandeld met mesothorium.* Nederl. Tijdschr. v. Geneesk. 2:2079-2083, 1914.

110. Mylius, K.: *Erfahrungen mit der Strahlenbehandlung tuberkulöser Erkrankungen des vorderen Augenabschnittes.* Ztschr. f. Augenheilk. 61:230-241, 1927.
111. New, G. B., and Benedict, W. L.: *Radium in the treatment of diseases of the eye and adnexa.* Am. Jour. Ophth. 3:244-250, (April) 1920.
112. Norton, A. B.: *Radium in eye diseases.* Homœop. Eye, Ear, and Throat Jour. 11:20-24, 1905.
113. Osmond, J. D.: *Roentgen therapy of acute infections of the antrum and frontal sinus.* Am. Jour. Roentgenol. 10:374-377, (Sept.) 1923.
114. Pagenstecher, H. E.: *Strahlenwirkung auf das fötale Auge. Experimentelle Untersuchungen über die Entstehung der Netzhautrosetten.* Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. 40:447-456, 1916.
115. Pardo, R.: *Dell'azione dei raggi di Röntgen sopra alcune affezioni oculari.* Arch. di ottol. 12:288-328, 1904-1905.
116. Park, F. E.: *Radium emanation in hay-fever.* Med. Rec. 93:284, (Feb. 16) 1918.
117. Paton, Leslie: *Case of posterior cataract commencing subsequent to prolonged exposure to X rays.* Tr. Ophth. Soc. U. Kingdom. 29:37-38, 1909.
118. Peter, Gustav.: *Glaucom nach Röntgenbestrahlung.* Strahlentherapie. 17:189-191, 1924.
119. Peter, L.: *Studien zur experimentellen Röntgen- und Radiumkatarakt.* Arch. f. Ophth. 125:428-462, (Dec.) 1930.
120. Pfahler, G. E.: *Roentgenotherapy in sarcoma of the orbit.* Jour. Am. Med. Assn. 84:87-93, (Jan. 10) 1925.
121. Pfahler, G. E.: *The treatment of cancer of the skin with X rays, radium and electrocoagulation and their special indications.* Med. Jour. and Rec. 128:261-263, (Sept. 19) 1928.
122. Politzer, G.: *Ueber den Einfluss der Röntgenstrahlen auf die embryonale Linse.* Wien. med. Wchnschr. 79:551-555, (April 20) 1929.
123. Pordes: *Ueber Röntgenbehandlung entzündlicher Erkrankungen.* Fortschr. a. d. Geb. d. Röntgenstrahlen. 35:23, 1926.
124. Pusey, W. A.: *Treatment of vernal conjunctivitis with radium. Technic.* Jour. Am. Med. Assn. 71:806-807, (Sept.) 1918.
125. Rados, A., and Schinz, H. R.: *Tierexperimentelle Untersuchungen über die Röntgenempfindlichkeit der einzelnen Tiele des Auges.* Arch. f. Ophth. 110:354-369, (Dec.) 1922.
126. Ramié, M.: *Cornée et rayons X.* Lyon, Thèse, 1922, 75 pp.
127. Ranzi: *Ein Fall von Rhinosklerom.* Fortschr. a. d. Geb. Röntgenstrahlen. 8:284, 1904.
128. Raynal, Albert: *Quelques essais de radiothérapie dans les affections chroniques de l'oreille moyenne et de l'oreille interne.* Jour. radiol. et d'électrol. 7:180-181, 1923.
129. Révész, V., and Kövari: *Die Röntgentherapie der Otosklerose.* Gyógyászat. 28, 1926.
130. Robinson, G. A.: *Radium therapy in diseases of the eye and adnexa.* Arch. Ophth. 55:328-337, (Feb.) 1926.
131. Robinson, G. A.: *Radium in postoperative treatment of polypoid sinusitis.* Arch. Physical Therap. 11:14-15, (Jan.) 1930.
132. Rohrschneider, W.: *Klinischer Beitrag zur Entstehung und Morphologie der Röntgenstrahlenkatarakt.* Klin. Monatsbl. f. Augenh. 81: 254-259, 1928.
133. Rohrschneider, Wilhelm: *Experimentelle Untersuchungen über die Veränderungen normaler Augenwebe nach Röntgenbestrahlung. I. Mitteilung. Zweck der Versuche. Definition der angewandten Röntgenstrahlung.* Arch. f. Ophth. 121:526-536, 1929.

134. Rohrschneider, Wilhelm: *Experimentelle Untersuchungen über die Veränderungen normaler Augengewebe nach Röntgenbestrahlung. II. Mitteilung. Allgemeines über Röntgenstrahlenwirkung. Veränderungen der Conjunctiva, Cornea und Uvea nach Röntgenbestrahlung.* Arch. f. Ophth. 121:537-559, 1929.

135. Rohrschneider, Wilhelm: *Experimentelle Untersuchungen über die Veränderungen normaler Augengewebe nach Röntgenbestrahlung. III. Mitteilung. Veränderungen der Linse, der Netzhaut und des Sehnerven nach Röntgenbestrahlung.* Arch. f. Ophth. 122:282-298, 1929.

136. Rohrschneider, Wilhelm: *Experimentelle Untersuchungen über die Veränderungen normaler Augengewebe nach Röntgenbestrahlung. IV. Mitteilung. Röntgenstrahlenempfindlichkeit der einzelnen Teile des Kaninchenauges bei einzeitiger Bestrahlung.* Arch. f. Ophth. 122:383-390, 1929.

137. Rohrschneider, Wilhelm: *Schädigung der Augen bei der Röntgenstrahlentherapie extraokularer Erkrankungen.* Deutsch. med. Wchnschr. 58:1126-1128, (July 15) 1932.

138. Rollet and Bussy: *Iritis nodulaire probablement tuberculeuse. Radiothérapie. Guérison.* Lyon méd. 129:457-458, 1920.

139. Rollet and Bussy: *Conjonctivite granuleuse grave. Radiothérapie. Amélioration.* Lyon méd. 129:458, 1920.

140. Sabbadini, D.: *Le modificazioni cliniche ed anatomo-pathologiche della congiuntiva trachomatosa in seguito al l'azione delle radiazioni secondarie.* Arch. di radiol. 2:584-688, 1926.

141. Scheerer, Richard: *Röntgenbestrahlung bei Uvealtuberkulose.* Klin. Monatsbl. f. Augenh. 75:27-61, 1925.

142. Schmidt, Karl: *Ein Versuch, den Heuschnupfen durch Röntgenstrahlen zu Beeflussen.* München. med. Wchnschr. 62:773, (June) 1915.

143. v. Schrötter, Hermann: Quoted by Weinberger, Maxmilian. Discussion. Wien. klin. Wchnschr. 17:1331, 1904.

144. Schwarz, Gottwald: *Die Röntgenstrahlen im Dienste der Ohrenheilkunde.* Monatschr. f. Ohren. 44:675-683, 1910.

145. Sgrosso, S.: *Contributo alla radioterapia delle affezioni oculari.* Arch. di. ottal. 29:305-319, 1922.

146. Sherer, J. W.: *Conjunctivitis from x-rays; incipient retinitis apparently due to the same cause: report of a case.* New York Med. Jour. 74:543-545, (Sept. 21) 1901.

147. Stephenson, Sydney, and Walsh, David: *On the curative treatment of trachoma by x-ray tube exposure and by high-frequency current.* Med. Press and Circular. 75:155-157, 1903.

148. Terrien, M. F.: *Radio-diagnostic et radiothérapie en ophtalmologie.* Arch. d'opht. 36:364-368, 1919.

149. Thiébault: *Notes et observations sur l'emploi du radium (épithéliomes, sclérites, trachome).* Clin. opht. 12:347-351, 1906.

150. Thielemann: *Experimentelle Röntgenschädigungen des Ohres.* Fortschr. a. d. Geb. d. Röntgenstrahlen. 37:563-564, 1928.

151. Thost, A.: *Die Behandlung von Erkrankungen der oberen Luftwege und der Ohren mit Röntgenstrahlen.* Monatschr. f. Ohrenh. 48:84-106, 1914.

152. Treutler: Discussion. Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. 32:338, 1905.

153. Tribondeau, L., and Belley, G.: *Cataracte expérimentale obtenue par röntgénisation de l'œil des animaux nouveau-nés.* Compt. rend. Soc. de biol. 63:126-128, (July 2) 1907.

154. Tribondeau, L., and Belley, G.: *Microphtalmie et modifications concomitantes de la rétine par röntgénisation de l'œil des animaux nouveau-nés.* Compt. rend. Soc. de biol. 63:128-130, (July 2) 1907.
155. Tribondeau, L., and Lafargue, P.: *Action différente des rayons X sur le crystallin des animaux jeunes et des animaux adultes.* Compt. rend. Soc. de biol. 63:716-717, (Dec. 3) 1907.
156. Tribondeau, L., and Larfargue, P.: *Étude expérimentale de l'action des rayons X sur la rétine et le nerf optique.* Compt. rend. Soc. de biol. 65:149-151, (July 7) 1908.
157. Tribondeau and Récamier: *Altérations des yeux et due squelette facial d'un chat nouveau-né par roentgénisation.* Compt. rend. Soc. de biol. 58:1031-1032, (June 6) 1905.
158. Urbantschitsch, Victor: *Über einige mit Radium behandelte Ohrenfälle.* Arch. f. Ohrenh. 96:15-21, (Aug.) 1915.
159. Uhthoff: Quoted by Darier, A.: *Die Anwendung des Radiums in der Augentherapie.* Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. 210-215, 1905.
160. Van Duyse: *Prolifération lymphomateuse hyaline de la conjonctive bulbaire droite. Guérison par les rayons X. Considérations sur quelques pseudoplasmes de la conjonctive.* Arch. d'opht. 25:402-421, 1905.
161. Viganò, E.: *Dacriociste e radioterapia.* Atti d. Cong. ital. di radiol. med. 1:233-236, 1913.
162. Viganò, E.: *I vantaggi della radioterapia nelle lesioni oculari consecutive a ferite e nei distacchi di retina.* Radiol. med. 7:223-229, 1920.
163. Wassing, Hans: *Beitrag zur Strahlenbehandlung tuberkulöser Augenerkrankungen.* Klin. Monatsbl. f. Augenh. 72:175-181, 1924.
164. Webster, J. H. D.: *X-ray treatment of two cases of otosclerosis.* Arch. Radiol. and Electroth. 26:69-75, (Aug.) 1921.
165. Wetterstand, G. A.: *Zur Kenntnis der Röntgentherapie bei Augentuberkulose.* Acta radiol. 7:639-645, 1926.
166. Wilkinson, Oscar: *Cataract probably due to x-ray exposure.* Am. Jour. Ophth. 3:435-436, (March) 1920.
167. Williams, C. H.: *A severe case of uveitis treated with radium.* Tr. Am. Ophth. Soc. 10:269-275, 1904.
168. Williams, F. H.: *Notes on radium: production of the gamma rays from the beta rays of radium: use of radium in some diseases of the eye.* Boston Med. and Surg. Jour. 150:559-561, (May) 1904.
169. Yocom, A. L., Jr.: *X-ray treatment of chronic otitis media.* Med. Herald. 44:268-269, (Nov.) 1925.
170. Yoel, M.: *Un nouveau cas d'ozène traité et guéri par le radium.* Arch. internat. de laryngol. 32:1001, 1926.
171. Zentmayer: Quoted by Ring, G. O.: *The value of x-rays in ocular therapeutics.* Jour. Am. Med. Assn. 47:994-1003, (Sept.) 1906.

Symposium on Physical Therapy

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ULTRAVIOLET AND INFRARED RAYS, HIGH FREQUENCY CURRENTS

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A BEAM of white light passed through a quartz prism is refracted and dispersed. These color bands thrown on a white screen show red at one end and violet at the other. This represents the visible part of the electromagnetic spectrum. A thermometer placed beyond the red end of this spectrum, where there is no visible light, will show a rise of the mercury. This is due to the heat produced by the invisible radiation known as infrared. The radiations with longer wave lengths than the infrared are known as Hertzian waves, used for radio transmission.

An unexposed photographic plate placed beyond the violet band of the visible spectrum, where there is no visible light, will show that in this region there is invisible radiation causing chemical changes in this photographic plate. This radiation of the electromagnetic spectrum is known as the ultraviolet.

The usual methods for classifying radiant energies is by their wave length or by their frequency. The unit of wave length is the angstrom unit, which is one ten-millionth of a millimeter. The visible spectrum is, roughly, between 4000 to 8000 angstroms.

The table (see next page) by Coblentz[1] gives a summary of the physical and physiologic characteristics of the infrared, visible and ultraviolet areas of the electromagnetic spectrum.

Sources of Ultraviolet Radiation. Not only is the sun regarded as the source of all energy, but it is also the best of all sources of radiation for ultraviolet therapy. However, owing to climatic conditions, sunlight is not always available for therapeutic use. Hence, if therapeutic ultraviolet radiation is desired, physicians are obliged

*Different Spectral Regions, Probable Depth of Penetration and Probable Physiologic Action of Rays from Different Sources**

Spectral Region	Penetration of Rays	Physiologic Action	Source
Far ultraviolet 1,800 to 2,900 A	Superficial 0.1 to 0.5 mm.	Photo-chemical	Metals in carbon arc and spark of metals (mercury arc)
Near ultraviolet 2,900 to 3,650 A	Superficial 0.5 to 1 mm.	Photo-chemical	Sun; metals in carbon arc; arc of metals
Visible spectrum 3,900 to 7,600 A	Superficial 1 to 5 mm.	Thermal; nerve stimulation	Sun; carbon arc
Near infrared 7,600 to 15,000 A	Deep 10 to 30 mm.	Thermal; nerve stimulation	Sun; carbon arc; gas-filled tungsten lamp
Far infrared 15,000-150,000 A	Superficial 3 to 0.1 mm.	Thermal; nerve stimulation	Carbon arc; infrared (radiant) heaters

* Wave lengths in angstroms, A.

to use some form of artificial radiation. The common artificial sources are the carbon arc, the hot mercury quartz arc, the cold quartz lamp, and the high frequency electrodeless discharge lamp.

The carbon arc is the hottest artificial source of ultraviolet radiation and in this respect is the closest approach to sunlight. Strandberg[2] of the Finsen Institute believes that in the treatment of laryngeal tuberculosis, if one is forced to use artificial energy instead of sunlight, one should choose a type of lamp which has a continuous spectrum and possesses the greatest amount of the longer ultraviolet, visible, and short infrared rays, rather than the shorter ultraviolet. This is the carbon arc.

The Council on Physical Therapy states that for tuberculosis of the bones and articulations, tuberculosis of the larynx and lymph nodes, the majority of authorities express a distinct preference for this type of generator. It should be remembered that the radiation from a carbon arc depends on (1) the size and kind of electrodes, (2) the direction of the current, and (3) the amount of electric current. Many carbon arc lamps sold to the public do not use large enough carbons or a sufficient amount of current to render them efficient.

The burner of the hot quartz lamp is an air-exhausted fused quartz tube containing mercury. There are two types: the air-cooled for general body irradiation and the water-cooled type for local use. The quartz tubes in these lamps gradually decrease in transparency and do not transmit as much radiation as when new. Therefore, erythema tests should be applied every three months to check the efficiency of the burner. The emission of this lamp contains little infrared, a fairly large amount of visible violet, and

a large amount of ultraviolet. The emission line at 2537 angstroms is practically of the same intensity as the lines at 3020 and 3130 angstroms, in contrast to the cold quartz lamp in which the line at 2537 angstroms contains over 95 per cent of the radiation emitted by the three lines.[1]

Most of our knowledge of ultraviolet radiation comes from the use of the carbon arc and the hot mercury quartz lamps. With the newer forms of lamps, few clinical reports with controls have been published.

The cold quartz lamp, according to Coblentz,[1] shows that of the total radiation of all wave lengths less than and including the line 3130 angstroms more than 95 per cent is contained in the resonance emission line of mercury vapor at 2537 angstroms. The erythemogenic efficiency of this lamp is high, but practically all this effect is produced by the line at 2537 angstroms. Coblentz calls attention to the fact that erythemogenic efficiency is not necessarily a criterion of its suitability for therapeutic purposes.

The electrodeless high frequency induction lamp is a round blub of quartz or Corex-D glass, without electrodes, surrounded by a high frequency coil connected to the transformer of a diathermy machine. The spectral energy distribution, the total ultraviolet output and the erythemogenic efficiency of this type of lamp, according to Coblentz,[1] were found to be nearly the same as that of the ordinary mercury arc lamp. Such a unit has proven useful in Kovács' clinic, but he believes that further reports as to its efficiency relative to the powerful new hot mercury arc are desirable.[3]

PHYSIOLOGIC EFFECTS OF ULTRAVIOLET RADIATION

Experimental and clinical data have shown that the spectral band of ultraviolet radiation of wave lengths shorter than about 3150 angstroms, occurring in sunlight and in some artificial sources of radiation, if sufficiently prolonged, has the power of creating vitamin D in the human body, raising the serum content of calcium and phosphorus, and promoting the fixation of these minerals in the body. Therefore, ultraviolet rays have a prophylactic and curative effect on rickets, infantile tetany or spasmophilia, and osteomalacia. This irradiation may also exert a beneficial action on other disorders of calcium metabolism, but the limits of such action, the conditions under which it may best be produced, and the specific action of the rays have not yet been fully explored.

The erythema producing effect of ultraviolet varies with the wave length of the ultraviolet radiation. It is maximal at 3000 and 2500 angstroms and shows a pronounced minimum at 2800 angstroms. The latent period and the reaction time is much shorter and the range of exposure time for producing an erythema without causing blistering is much greater for the 2500 angstrom area than for the longer wave lengths.

This erythema is an inflammatory reaction. With a mild erythema there is a dilatation of the capillaries, an increased blood flow, and a marked leucocytosis in the irradiated area, followed by a restoration to normal. This reaction first occurs within one to six hours, depending on the intensity of the radiation and the erythemal sensibility of the skin. The reaction usually subsides in one or two days. With a severe erythema dose there is often a serofibrinous and hemorrhagic exudate and even thrombi in the blood vessels of the skin, with degeneration and necrosis of the skin. It has been shown that a moderate erythema of the skin is associated with a local increased bacteriolytic and bacteriophagic power of the blood serum. This fact and the local increase of leucocytes may be the explanation of the favorable action of an erythema dose of ultraviolet radiation in the treatment of erysipelas.

The Council on Physical Therapy[4] has adopted and, until a more practical procedure is proposed, will use the erythemal reaction as a basis for judging the effectiveness of ultraviolet lamps for three important reasons: (1) it is a physiologic reaction that has been established with a high degree of accuracy, (2) in case of exposure to intense sources of ultraviolet radiation it is a simple and practical means of preventing severe burns, and (3) in case of weak sources of ultraviolet radiation it is an efficient safeguard against lamps that are deficient in ultraviolet radiation. The Council's specifications of minimum intensity are such that at 24 inches from the front edge of the reflector of the lamp to the skin, the time of exposure to produce a minimum preceptible erythema (one that disappears in less than 24 hours) will not be longer than 15 minutes for a therapeutic lamp and 60 minutes for the so-called home sun-lamps.

Pigmentation of the skin and erythema, although associated, are not interdependent. Pigmentation is produced by longer wave lengths and may be produced by other agents. The function of pigmentation is still a matter of controversy, and will not be considered here.[3]

The bactericidal effect of ultraviolet radiation is exerted by radiation from 3000 to 2500 angstroms. Unfortunately, there are many differences in destroying bacteria in a laboratory test tube and in the human body. First, ultraviolet radiation has only a skin penetration of from 0.1 to 1 mm. Second, it has not been shown that one kind of protoplasm is more resistant to ultraviolet radiation than another. Therefore, in the human body, there is no possibility of a direct bactericidal effect in the tissues by ultraviolet radiation and when the bacteria are on the surface it is a probability that the sensitiveness of the invading microörganisms and the living human tissues are the same. So that it would probably be impossible to destroy the bacteria without destroying the tissues. These facts would seem to indicate that local ultraviolet radiation in sinusitis or chronic otorrhea is limited by its inability to reach the affected area, its inability to penetrate even a small surface accumulation of pus, and that the dosage of radiation sufficient for bacterial destruction on the surface causes normal tissue necrosis.

In superficial wounds the erythemal and bactericidal actions of ultraviolet radiation have definite benefits. Apparently these are not the only beneficial effects of radiation as in the treatment of superficial wounds the benefits of radiation are greater if the wounds are exposed not only to the ultraviolet but also to luminous and infrared rays, which are so preponderatingly present in natural sunshine and carbon arc radiation, than if only exposed to the shorter ultraviolet rays of the mercury arc.[5]

General ultraviolet radiation may have a slight therapeutic influence on secondary anemia, but such influence appears to be limited. There is much disagreement regarding the effects on the blood of general body ultraviolet radiation.

The Council on Physical Therapy still believes, that as far as normal persons are concerned, the claim that exposure to ultraviolet rays increases or improves the tone of the tissues or of the body as a whole, stimulates metabolism, or tends to prevent colds has not been clearly substantiated.[4]

Oft-repeated exposures to ultraviolet radiation over long periods in persons who have a low tolerance to ultraviolet rays may lead to degenerative changes in the skin. Grossly excessive exposures of the entire body may in certain cases cause serious illness or even death. Grossly excessive exposures to a local area may in some cases lead to permanent deleterious changes in the skin.

INFRARED RADIATION

Beyond the red of the visible spectrum is the infrared radiation. Infrared rays are the heat rays we feel when standing near a radiator or a stove. The spectral emission and the total intensity of the infrared rays from an ordinary room heater is essentially the same as that of a therapeutic infrared generator.

Infrared radiation from a luminous source penetrates subcutaneous tissues from 10 to 30 mm. These rays are known as near or short wave infrared. Sonne[6] found that during the application of incandescent radiation of maximum skin tolerance the surface temperature of the skin rose to 43.8° C. and the subcutaneous temperature at a depth of 0.5 cm. was 47.7° C. Far infrared radiation from a heater consisting of an electrically-heated solid rod, or a resistance wire embedded in or wound on an electrically nonconducting refractory material, penetrates only 0.1 mm. to 3 mm. Sonne noted on application of this form of infrared from the so-called infrared generators that the skin temperature was 45.5° C. and the subcutaneous temperature at 0.5 cm. was 41.7° C. This means that the infrared radiation from an incandescent bulb at maximum skin tolerance is more effective in increasing the subcutaneous temperatures than the radiation from an infrared heater at the same tolerance. These long wave infrared radiations exert their maximum heating effects on the superficial area.

The physiologic effects of infrared radiation are local and general. The local effects are an action on vasomotor and sensory nervous systems of the skin. The local effects of heat have been well shown in a motion picture made for the Council on Physical Therapy by Clark. This picture shows the blood vessels of a rabbit's ear in a transparent chamber viewed through a microscope. With a moderate amount of heat the capillaries are dilated, the circulation is increased, a local increase in leucocytes, and a sticking of the leucocytes to the vessel walls. With a moderate amount of heat, tissue metabolism is accelerated and the rate of exchange between the blood and tissues is increased and reaches its optimum so that blood entering the vein contains 60 to 65 per cent of its saturated value of oxygen, as compared to 95 per cent at the arterial end of the capillary. Above 102° F., the rate of blood flow may become so rapid that the blood at the venous end of the capillary contains 91 per cent of its saturated value of oxygen.

The rapid circulation increases the thermal conductivity of the tissues and distributes the heat through the body and thus

hinders the rise in local temperature. The changes are due partly to nervous reflexes and consequently may involve distant areas not primarily affected by the temperature.

When heat is applied to an area, the superficial capillaries and veins are dilated, the blood flow is increased and the capillary pressure is raised. The hypothesis, according to Bazett,[7] is that fluid interchange between the blood and the tissue fluids depends normally on a simple balance between the hydrostatic pressure of the blood in the capillaries and the osmotic pressure of the protein colloids, which enables them to pass the capillary wall and exert an attractive force on watery solutions of diffusible substances. A rise of surface temperature that increases the local circulation rate and raises capillary pressure will increase the rate of edema formation when the venous pressure is maintained at a constant level. Therefore, a rise of temperature must be expected to increase lymphatic drainage greatly, and this may be the explanation for the relief of pain when heat is applied in acute or chronic sinusitis. When lymphatic drainage is obstructed the collection of fluid might cause pressure on venous channels, and so, secondarily, interfere with circulation. This may be the cause of the increase of pain occasionally seen when heat is applied.

Bazett states that when blood is warmed the dissociation constants of the acid radicals of the proteins are increased to a much greater extent than is that of carbonic acid. In consequence, if blood is warmed and the total carbon dioxide content is constant, the proteins combine with much base previously in the form of sodium or potassium bicarbonate, the carbon dioxide thus freed is added to the free carbonic acid originally present, the acidity is greatly increased, and the carbon dioxide tension is raised. The change in carbon dioxide tension is further exaggerated by diminution in its solubility. An alteration of 1.8° F. causes an increase of acidity comparable to the normal difference between arterial and venous blood. In spite of these changes the direct effect of the temperature on the blood far outbalances them so that the changes in acidity are mainly determined by the latter factor. Bazett's figures on this subject show that the great changes in gas tension and acidity may be of great importance in modifying the reactions of tissues to infections. With a surface temperature of 59° F. and a blood temperature of 62.6° F., the carbon dioxide tension was between 15 and 20 mm. and the oxygen tension between 15 and 30 mm. while with a surface temperature of 107.6° F. and a blood

temperature of 104° F., the carbon dioxide tension was 45 mm. and the oxygen tension between 70 and 95 mm.

Bazett states that the activity of leucocytes in phagocytosis is probably greatly modified by temperature. The locomotion of leucocytes in vitro is certainly so affected; the maximal velocity is reached at 104° F.

Any change in temperature has a profound effect on the metabolic changes in any tissue. Where this effect may be observed on isolated organs, the change is such that these chemical changes are increased two or three times for a rise of 18° F.

Many of these changes are dependent on a rise of temperature that apparently is difficult to secure in nasal conditions. The advantages of infrared radiations over the methods of conductive heating are (1) its action extends to a much greater depth, (2) there is no pressure over the parts treated, and (3) the parts may be kept under constant observation without difficulty.

HIGH FREQUENCY CURRENTS

Medical diathermy is the production of heat in the body tissues for the therapeutic purposes by high frequency currents, insufficient in amount, however, to produce temperatures high enough to destroy the tissues or impair their vitality. These currents are applied locally by three methods: (1) contact metal electrodes, known as conventional diathermy; (2) with a high frequency electric field; and (3) with a high frequency electromagnetic field.

The first method is the one by which conventional diathermy is applied. The frequency of oscillations is usually from one-half million to two million cycles per second.

The second method is the use of the high frequency electric field. The frequency of oscillations may be from ten million to one hundred million cycles per second. By the electric field method, it is assumed that the heat is due to the ohmic resistance of tissues to high frequency alternating currents passing through them; or again, it may be due to dielectric losses within the tissues in the electric field; others believe that it may be a combination of the two effects.

The third method of heating tissues by high frequency currents is by electromagnetic fields or so-called electromagnetic induction. The exact physical phenomena taking place within the tissues, causing generation of heat have not been fully explained. However, it is believed that within the helical coil, through which

the high frequency current flows, there is set up an alternating flux having the same frequency as the current within the coil. If a conductive material is placed within the coil an electromotive force will be induced in it. As a result of this induced voltage, eddy currents of the same frequency as the exciting current will flow in the conductive material, generating heat in the tissues.

Physiologic Action of Medical Diathermy. There are certain special claims made for the physiologic action of these currents. (1) Special selective thermal action, (2) special biologic action, (3) specific bactericidal action, and (4) a more effective and less dangerous method of heating human tissues than conventional diathermy.

Special Selective Thermal Action. We have made 291 in vivo experiments in tissue heating. These experiments were made on adult male medical students averaging in weight 170 pounds. Our procedure was to introduce a hard rubber canula with trocar into the midpoint of the thigh through the quadriceps muscles for a depth of two inches or until the femur was encountered. The diameter of the canula was just large enough to introduce a 16 gauge hyperdermic needle. The trocar was removed and replaced by a thermocouple made of constanton and copper wires (28 gauge Leeds and Northrup double cotton covered and enameled), soldered into the tip of a 16 gauge lumbar puncture hyperdemic needle. The thermocouple was connected to a potentiometer (Leeds and Northrup portable precision type). The couple was calibrated against a Bureau of Standards calibrated thermometer with scale divisions in one-tenth degree F. The thermocouple was removed from the field during the passage of the high frequency current. To conform to clinical practice, the application of current was made for twenty minutes' duration. Temperature readings were made at the beginning and termination of the treatment.

For these tests we have used practically every machine in operation today. The technical application was made by expert technicians and supervised by an engineer from the factory of the particular machine used. The machines, therefore, were operated under the most favorable conditions. The technics used were those advocated by the manufacturers. We did not wish to make these experiments a purely laboratory procedure but rather to conform to ideal clinical practice.

Our applications were made as follows:

1. *The high frequency electric field*
   a. Condenser pad electrodes
   b. Air gap electrodes of varying types
   c. Double cuff electrodes

2. *The Electromagnetic field*
   a. Coil technic
   b. Flat pancake coil or disk

We employed the following wave lengths: 6, 8, 9, 12, 14, 15, 18, 23, 24, and 25 meters.

Condenser pads were applied laterally on either side of the thigh with the canula centered between them. This is known as the "through and through" technic.

There was a great deal of variation in the materials of the so-called air gap electrodes. Some used bare metal encased in glass containers, others used metal encased in rubber with air as the dielectric between skin and electrodes, while others used sponge rubber, solely, for the dielectric. Many variations in the method of application of the electrodes were made.

Double cuff electrodes were applied around the thigh equidistant from the canula above and below. Here again there were differences in the width of the cuffs. Each manufacturer supplied the cuffs he advocated for his particular generator.

In the electromagnetic field there was greater uniformity both as to the cable itself and the number of turns employed. Three or four turns of the cable were applied around the thigh with the canula midway between the extremities of the coil. The disk was a cable in a flat pancake form embedded in a hard rubber container. The cable formed into a pancake coil without the container was also used and applied so that the canula was in the center of the innermost turn.

From these experiments

1. We believe that wave lengths between 6 and 25 meters are not a marked factor in tissue heating in the living subject, but that differences in machines, the energy delivered to the patient and technic play important roles.
2. The electromagnetic field produces effective heating of live human tissues.

3. The double cuff method of the electric field is an effective technic.
4. Air spaced electrodes are effective for deep tissue heating provided the proper application is used.

In other articles on this subject we have reviewed the literature, and will not consider it here.

Specific Biologic Action. Mortimer and Osborne,[8] in reviewing the literature, believe that there is no conclusive evidence from the literature nor were they able to substantiate in their investigations the claim of specific biologic action of high frequency currents (short wave diathermy). In their opinion the burden of proof still lies on those who claim any biologic action of these currents other than heat production.

Eidinow[9] states that in his experiments he failed to demonstrate any direct bactericidal action or any biologic effect apart from that due to heat.

It is believed that certain previous experiments showed that the therapeutic effects of short wave currents are limited to the effects of heat produced. This view is supported by the experimental work of Malov.[10]

Hill and Taylor's experimenting with high frequency currents on an excised heart immersed in a small quartz vessel containing Ringer's solution, showed that the behavior of a frog heart, cilia and nerve muscle preparation exposed to the 3.4-meter wave length is exactly the same as when merely heated in Ringer's solution. These authors conclude that the biologic effect is due to heat.[11]

Wetzel and Kiesselbach,[12] in their work with wave lengths of 12 and 8 meters, observed the effect of heat on tadpoles. The tests according to Wetzel and Kiesselbach support the assumption that the chief cause of the biologic action of the short waves is the heat that is produced by the energy exchange.

Curtis, Dickens and Evans,[13] in a recent article summarizing their investigations on specificity state:

"If such an effect exists, it should be possible for the discoverers to describe at least one clear-cut experiment which could be repeated by other workers. In the absence of such evidence we consider that the great mass of inconclusive observations which has been presented is a very insecure foundation for the rapidly growing belief in specific short wave therapy. Whilst the possible existence of specific actions of ultra short waves cannot be denied,

in our opinion such effects have not as yet been adequately demonstrated. We therefore find ourselves in agreement with the conclusions of a recent report to the Council on Physical Therapy of the American Medical Association, by Mortimer and Osborne: 'There is no conclusive evidence from the literature; nor were we able to substantiate the claim of specific biologic action of high frequency currents (short wave diathermy). In our opinion the burden of proof still lies on those who claim any biologic action of these currents other than heat.' "

Kovács[14] states that claims for selective heating by short wave diathermy in laboratory experiments have not been corroborated in the living human organism and do not hold out hope for any practical therapeutic value.

Specific Bactericidal Action. Mortimer and Osborne reviewed the literature on this subject and in their own experiments they subjected broth cultures of staphylococci, streptococci, B. melitensis, gonococci, meningococci, and B. typhosus to a 6-meter wave for twenty minutes, during which time the temperature of the culture rose to 40° C. without observing any change in the growth of the microörganisms. They subjected rats with experimental pneumonia for three minutes daily to the 6-meter wave without observing any change in the fatal course.

The experimental work that claims specific bactericidal action for these high frequency currents may be more rationally explained, according to Mortimer and Osborne, on the basis of "point heating," which raises the temperature of the microörganisms above their thermal death point without a corresponding elevation in the temperature of the medium. It still remains to be demonstrated whether such test tube results can be secured with infection in the body.

At the clinic at Northwestern University Medical School, studies of radiant heat by means of infrared rays and of conduction heat by means of an electric resistance heating pad applied to the eye and orbit in narcotized dogs were made for comparison with conventional diathermy. Now studies are being made with short wave medical diathermy. These show that in the orbit with a heating pad there is a rise of 1.31° C., with infrared radiation 3.25° C., and with diathermy 4.49° C.; in the cavum oculi, heating pad 2.29° C., infrared radiation 3.02° C., and diathermy 6.98° C.; and in the anterior chamber, heating pad 2.64° C., infrared 4.47° C., and diathermy 6.54° C.[15]

A comparison of these results demonstrates that the skin and superficial tissues tolerate the diathermy currents and the short infrared radiation better than the conductive heat from the electric resistance heating pad, so that a greater input of energy and consequently a greater rise of temperature is produced by diathermy than by either of the other two methods. Our experiments now in progress would indicate that the skin and superficial tissues tolerate short wave diathermy better than conventional diathermy, and a greater rise in temperature is produced. It is significant that even in the anterior zones the heating power of conventional diathermy within the limits of skin tolerance is greater about 2° C. than infrared radiation and much greater about 4° C. than the heating pad.

Andreen and Osborne[16] made measurements of the temperature of the maxillary sinus after treatment by various methods of heating. With short wave diathermy and infrared there was an average rise of 0.9° F. These readings agree essentially with Gale,[17] who secured 0.5° F. rise, and Tebbutt,[18] who found no change or a drop in temperature. Rossenwasser and Burman[19] with short wave electric field, secured an average rise of 1.31° F. With these small rises of temperature in the antrum, it would seem that short wave medical diathermy would have an action only on one wall, giving a local circulatory increase.

At the present time it is believed that the local physiologic effects of the three methods of applying medical diathermy are limited to the effects of the heat produced. These effects were given under infrared irradiation. The advantage of this form of heating is the penetration in certain cases, for instance, in the orbit there is much more heat produced by diathermy than with infrared. Its disadvantages are that the generation of heat within the tissues can only be judged if experiments are performed to determine it. In the antrum, there is little heat produced and no more than with infrared. Secondly, the regulation of dosage with short wave diathermy is an even more empirical procedure than with ordinary diathermy. The milliameter of the high frequency machine is connected to the oscillatory circuit and serves only as an indicator that electrical energy is passing and does not indicate that the energy is passing through the patient. Third, unless careful technic is used the generation of heat in the tissues is less with short wave diathermy than with conventional diathermy. Fourth, there are many inefficient machines on the market. Both of these latter disadvantages can be avoided by reading the published ac-

ceptances of short wave diathermy machines by the Council on Physical Therapy. The Council only accepts short wave machines that prove more efficient for deep tissue heating by using the tests as given on the human thigh than long wave diathermy machines, the results of these tests are given, which permit comparison with other machines, and the effective technics are also given.

The advantages of short wave diathermy are that it is by far, with correct technics and an efficient machine, the best method for producing heat in the depth of human tissue. Second, it is possible to produce burns, but the danger is less with short wave than with conventional diathermy.

SURGICAL APPLICATION OF HIGH FREQUENCY CURRENTS

There are three common methods for using high frequency currents for the destruction of new growths and diseased tissue and for cutting through living tissue with minimal bleeding.

Electrodesiccation is performed with a needle-point electrode connected to a single terminal of a high frequency machine and held in contact or at a slight distance from the part to be treated. The tissue cells are shrunken and shriveled, the nuclei condensed, the blood vessels are thrombosed, and there was no hemorrhage.

Electrocoagulation is done by two technics. The usual method for destruction of new growths is to connect a needle or small ball or disc electrode to one terminal of the high frequency machine and a large dispersing electrode to another terminal of the machine. There is a widespread coagulation produced at the active electrode. Another method used in tonsil coagulation is an electrode with two needle-point electrodes about one-fourth inch apart in an insulated handle with the wires from the needles connected to separate terminals of the high frequency machine. Coagulation occurs in the area between these points.

Electrocoagulation produces a grayish white discoloration of a varying depth. Histologically, the cells are fused in a structureless homogeneous mass with an appearance like hyalinization. Ellis[20] notes in this coagulated area two zones. The inner zone shows a typical coagulation necrosis like the necrosis of ordinary skin burns. In the section there is an outer zone of shriveled cells and dilated blood vessels which in a few days becomes infiltrated with round cells, fibroblasts, and giant cells. Ellis points out that in coagulation of tissues, the specific resistance of tissues varies tremendously, e.g., fat is more than eight times as resistant as muscle.

Therefore, even the inner zone of complete coagulation necrosis is always uneven according to the variation of resistance in different tissues. The resulting injury in electrocoagulation is unpredictable. This accounts for the hemorrhages sometimes seen after electrocoagulation.

The third method for the surgical application of high frequency current is the cutting current. Here a needle or a thin blade is connected to one terminal of a high frequency apparatus, producing a current of undamped oscillations. The histological picture shows an inner zone of disruption of the cells and an outer zone showing elongation of tissue cells and a slight amount of coagulation. The width of these zones with a fast cutting current is about 0.1 mm. Ellis found that only 60 per cent of the cuts produced in dog's skin healed by primary intention.

CONCLUSION

1. Karsner and Goldblatt have suggested in an article for the Council on Physical Therapy of the American Medical Association that the evaluation of methods used in physical therapy must not be measured by the opinions of the physician, but rather by the facts he can demonstrate. Such critical studies were made on the use of ultraviolet radiation in the treatment of rickets.
2. Many of the physiologic actions of infrared and ultraviolet radiation and high frequency currents are known, but their effects have not been criticially evaluated. Therefore, these agents should be used only as adjuncts to other forms of treatment.

BIBLIOGRAPHY

1. Coblentz, W. W.: J.A.M.A., 103:3:183, July 21, 1934, and 103:4:254, July 28, 1934.
2. Strandberg, Ove.: J.A.M.A., 90:20:1595, May 19, 1928, and Lancet, ccxxvi:5760:128, Jan. 20, 1934.
3. Kovács, Richard: *Electrotherapy and Light Therapy*, 2nd Ed., Phila., Lea & Febiger, 1935.
4. Handbook of Physical Therapy, 2nd Ed., 1936, Am. Med. Assn.
5. Laurens, Henry: J.A.M.A., 103:19:1447, Nov. 10, 1934.
6. Sonne, C.: Arch. Phys. Therapy, 10:93, June, 1929.

7. Bazett, H. C.: *Principles and Practice of Physical Therapy,* Hagerstown, Md., W. F. Prior Co., 1:4, 1934.
8. Mortimer, B., and Osborne, S. L.: J.A.M.A., 104:1413-1419, April 20, 1935.
9. Eidinow, A.: Proc. Roy. Soc. Med., 28:307, Jan., 1935.
10. Mavlov, N. N.: Strahlentherap. 53:326, 1935.
11. Hill, Sir Leonard, and Taylor, H. J.: Lancet, 1:6:311, Feb. 8, 1936.
12. Wetzel, G. W., and Kiesselbach, A.: Deutsch. med. Wehnsch., 18:725, May 1, 1936.
13. Curtis, W. E., Dickens, F., and Evans, S. F.: Nature, 138:63, July 11, 1936.
14. Kovács, Richard: Arch. Phys. Therapy, 17:7:432, July, 1936.
15. Moncrief, W. F., Coulter, J. S., and Homquist, H. J.: Am. J. Ophth., 15:3, March, 1932; and 16:3, March, 1933.
16. Andreen, M. A., and Osborne, S. L.: Arch. Otolaryn., 24:331-337, Sept., 1936.
17. Gale, C. K.: Laryngoscope, 45:520, July, 1935.
18. Tebbutt, H. K., Jr.: Arch. Otolaryng., 22:733, Dec., 1935.
19. Rosenwasser, H., and Bierman, W.: Arch. Otolaryng., 25:5:555, May, 1937.
20. Ellis, J. H.: *Principles and Practice of Physical Therapy,* Hagerstown, Md., W. F. Prior Co., II:19, 1934.

Symposium on Physical Therapy

PHYSICAL THERAPY IN OPHTHALMOLOGIC PRACTICE

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WITHIN the time at my disposal it will be possible to consider only a few phases of physical therapy and my remarks will be limited to procedures which have, or seem destined to have, a definite place in ophthalmologic practice.

PHOTOTHERAPY

This subject may be considered very briefly, since few recent advances in it have been made. The indications for general *phototherapy* are well-known, although ophthalmologists are apt to forget them. The object of treatment to produce repeated mild skin reactions, with resulting increase in various bactericidal constituents of the blood, and better utilization of calcium and vitamin D by the tissues. In phlyctenulosis such treatment is nearly always indicated when proper use of sunlight cannot be secured. Using a large mercury vapour or carbon arc, one-third of the body surface is irradiated at a sitting, the dose being chosen which will produce a slight erythema. Such treatments are given to other areas twice a week and repeated until 15 to 25 treatments have been given. Administration of calcium and vitamin D by mouth is a useful adjuvant to general phototherapy. In any form of ocular inflammation affecting persons with poor general resistance, and especially in cases of uveitis considered to be tuberculous, general phototherapy may be of considerable value and should be more often employed. The same is true of that severe and intractable condition, sclerosing keratitis.

Local Phototherapy has not lived up to the expectations which were held for it fifteen years ago. A few ophthalmologists still

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uphold its efficacy in severe corneal infections, such as serpent ulcer. Many, on the other hand, get along without it entirely, while others employ it in a relatively small number of cases, chiefly of superficial corneal infection. At the 1928 Academy meeting, in a resumé of phototherapy, I gave reasons for discarding the procedure in serpent ulcer, other deep corneal infections and inflammation of the posterior segment. On account of the complete absorption of ultra-violet rays by a very thin layer of opaque tissue, it seemed that its only possible effect could be expected in superficial forms of keratitis such as dendritic or herpetic ulcer, degenerative ulcers on the surface of old scars and the ulcers of trachoma. For many years I treated all ulcers of this type with the Birch-Hirschfeld Uveol Lamp or with a home-made arc-lamp embodying quartz lenses and a filter of Corning Glass No. 958 A. With the former lamp, treatments of 5 to 8 minutes were given daily, since even with such long exposures erythema is not produced. With the latter lamp erythema is produced in about one minute, so treatments were limited to 50 seconds and given three times a week. The lesions were stained with fluorescein or 1 per cent Rose Bengal before each treatment. A distinct impression was obtained that the light was of value in such cases, since ulcers usually healed after two or three and occasionally after one such treatment. Other agents, however, such as iodine, were usually employed, so that healing could not with certainty be attributed to the light. Also there were a few cases which did not heal on such treatment but required other measures. One of these measures, roentgentherapy, will be discussed later. Experimentally, Gunderson could demonstrate no effect of ultra-violet light on the herpes virus. Since most of these superficial ulcers heal on the use of chemical agents and since, of those which do not, results have apparently been obtained by roentgentherapy in cases which resisted phototherapy, the useful field for local phototherapy seems to be contracting further and further. It is probably not too much to say that the ophthalmologist who is familiar with the use of other agents, especially roentgentherapy in suitable cases, can dispense with local phototherapy entirely.

USE OF COLD

I hesitate to insult the intelligence of this body by speaking of such a rudimentary subject as the use of cold, but would like to report something which it took me ten years to learn for the benefit of a few who may have overlooked it. This is the tremendous value

of cold when applied after certain operations, and a simple but effective way of applying it. Anyone who will compare alternate operations for squint which are treated with and without or, as I did, the results of ten years without and ten years with routine use of cold, can hardly fail to be impressed with the difference in post-operative reaction. Chemosis and congestion after squint operations, with the use of cold, are minimal and in many cases almost entirely absent. The effect is even more striking following simple evisceration of the globe, in which, without the use of cold, chemosis was always extreme and very persistent. In ptosis operations, since using Friedenwald's procedure of protecting the cornea by the lower lid, it has become possible to use cold, and the results are well worth the trouble. The practical method of applying cold is that used for Dr. Loyal Davis' neuro-surgical cases at Passavant Hospital and is probably familiar to most physicians. It is the use of finely chopped ice in a thin rubber glove, the fingers of which are tied off so that a small flexible bag is formed. This is covered by a single layer of thin gauze and applied to the closed lids, which may be covered by a single layer of sterile gauze if skin sutures have been employed. It is used during the alternate waking hours, beginning immediately after operation and continuing for three to four days, as a rule. I have never seen any evidence of delayed healing or of damage to the corneal nutrition resulting from such use of cold. The effect on post-operative pain, as well as on chemosis, is often gratifying.

THERMAL THERAPY

Infra-red Lamps. Dr. Coulter has given experimental evidence indicating that greater heat may be produced in the tissues with infra-red lamps than with heating pads, and almost the same superiority probably holds when infra-red heat is compared with moist hot packs. There are also practical reasons for preferring infra-red lamps for routine hospital or home use. Such heat is clean and may be employed after operations without danger of infection. It avoids direct contact with the lids, which is of advantage in recent operations or wounds. The use of lamps lessens the amount of nursing care required, as compared with moist-heat, since patients are easily taught to adjust the lamp themselves to the limit of skin tolerance. The more expensive infra-red lamps which are usually available in hospitals may be replaced by very inexpensive models for use by patients at home.

The indications for infra-red therapy include all forms of inflammation of the eyes and adnexa except those in which danger of hemorrhage exists. Other exceptions are glaucoma and the early stages of conjunctivitis in which swelling and chemosis might be increased. In acute iritis, post-operative iritis, acute cellulitis, and deep infection of the lids heat is especially important and may be used almost continuously.

Heat generated by electrical energy in the tissues will be discussed only in its non-surgical aspects. We must admit, with Coulter, that the only effect of both long- or short-wave diathermy is that of heat and need not involve any mysterious electrical phenomena to explain the effect observed. Reports on the use of *local non-surgical long-wave diathermy* in ocular diseases have been numerous and a variety of conditions have been treated, as shown in a recent review by Nugent.[2] Dr. Coulter has compared the efficacy of long-wave diathermy with that of short-wave therapy. He has shown that with both methods high temperatures can be produced in the deeper tissues of the eye and orbit than is possible with infra-red lamps. With the short-wave method the higher temperatures may be reached more quickly and with less danger of burning the skin. With the glass electrodes now in use the effect may be fairly well localized. It is not known exactly what temperatures are tolerated by the normal ocular tissues without producing permanent degenerative changes. Hoffman and Kunz,[3] using long-wave diathermy, found that at 43.5° C., or 110.3° F., marked circulatory changes occurred in the choroid, including hemorrhages. At 42-43° C. (107.6° to 109.6° F.) the retinal ganglion cells showed edema and degenerative changes. Lens opacities were seen only with somewhat higher temperatures after one exposure, but the effect of repeated exposures was not determined. The duration of Hoffman's experiment was 15 to 25 minutes, the maximal temperature being maintained for ten minutes.

It would seem unwise to raise the temperature inside the eye much above 105° F. (40.5° C.) since the danger of hemorrhage must be considered as probably greater in eyes with inflammatory conditions or disease of the blood-vessels.

The difficulty in obtaining a dosage which will assure this limit of temperature is at once evident. All that can be done is to imitate closely the technique with which the desirable temperature has been obtained in experimental work, and that which has been employed in a large number of cases by reliable observers without

causing detectable damage to the eye. Kokott[4] employed the Siemens Ultratherm generating waves of 6 meters, the electrodes being placed in contact with the skin 2 cm. apart. He employed the technique recommended by the manufacturers for therapeutic work. The highest temperatures were obtained in the vitreous, which increased 4.8° C. in ten minutes and only slightly more after 15 minutes.

This temperature would approximate 107.2° F. Heating was less in the orbit, averaging 2.9° C. to 4.8° C. in the vitreous. There was some variation in the temperatures produced in various animals. He believes exposures of ten minutes are long enough to produce a practical maximum heating without dangers of longer exposures, especially that of producing cerebral disturbances. He obtained some burns with this technique and advises that more exact dosage and localization of the current is desirable.

Puntenney and Osborn, in some work now being conducted at Northwestern University, which will be published later, obtained results which agreed in most essentials with those of Kokott. With the same Siemens instrument, the glass electrodes being placed one over the eye and the other back of the neck, the temperature in the vitreous was raised within twenty minutes to 107-109° F. Cooling from the highest point occurred rapidly after the current was turned off, the temperature reaching 103-104° F. within twenty minutes and usually returning to normal within one hour. The body temperature was considerably higher than in Kokott's experiment but in most animals it remained an average of 3° F. lower than that of the vitreous. When the two electrodes were placed on either side of the eye such high temperatures were not obtained, the maximum being 106.7° F. and the average 103° F. Cooling also occurred much more quickly with this technique. With a ten minute exposure almost the same temperature could be reached but cooling was much more rapid. Careful measurement of the retinal vessels by Lambert's method revealed no actual dilation of the larger vessels in any animal, which is in contradiction with the impression gained by many observers from their ophthalmoscopic findings. This method is very accurate, certainly more so than the usual impression obtained from the ophthalmoscopic picture. It does not exclude, of course, dilation in the capillary bed. Individual differences between animals were considerable and it certainly cannot be stated that the same temperatures could be produced in man by approximately the same technique so that direct observation on human eyes before enucleation is desirable and is being

undertaken. The temperatures here recorded, while higher than some of those with which Hoffman observed damage to retinal structures, were not maintained so long as in his experiments.

Of clinical reports, that of Gutsch[5] gives his experience in treating 276 cases of ocular disease. He employed the same machine as did Kokott using glass shell electrodes, held 1 to 3 cm. from the globe with exposures beginning at 6 to 8 minutes and being increased to 15 minutes in certain cases. Treatments were given two to three times a week. He saw no evidence of damage produced in any case. He saw no definite beneficial effect in corneal diseases, glaucoma or scleritis, and the effects in acute iritis were doubtful. He was impressed by the benefits obtained in cases of chronic iridocyclitis, especially the tuberculous form, and in vascular diseases of the fundus. In three cases of conglomerate tubercle of the iris and two of the choroid the lesions disappeared rapidly under treatment. In acute orbital cellulitis and acute dacryocystitis the inflammation subsided rapidly. Wegner in discussing Gutsch's paper, reported no results in chronic uveitis and states that he saw a retinal hemorrhage occur after treatment. Scheyling[6] employed a similar technique and reports good results in various inflammatory conditions, but gives few details. Brief clinical reports by Carlotti[7] and Kuwahara[8] also claim good results for the method. The report of Krause[9] is chiefly clinical but includes observation of rabbits treated for ten successive days with the usual dosage. No damage to the eyes was noted. Best clinical results were obtained in infection of the lids and dacryocystitis in which conditions resolution occurred with striking rapidity. One case of post-operative iritis with hypopyon cleared up rapidly under treatment. No results were seen in sympathetic ophthalmia, or disciform keratitis. He reviews the observations of de Decker and Arendt, Grüter, Sattler and Bartels from which it is difficult to draw conclusions since the best results were in herpetic keratitis of the superficial variety, which often heals rapidly on other treatment. Results in interstitial keratitis were not definite. Kiewe[10] recorded the temperatures produced by various methods but details are not given in the available reports of his work.

A number of observers were convinced that dilatation of the retinal vessels could be seen following treatment, but no accurate measurements were made.

In this country, Walker[11] has employed short-wave diathermy in patients with vitreous opacities following detachment operations and due to other causes. He employs an apparatus delivering 1500

ma. with small oval electrodes suspended over the eye. He uses 15 minute applications with the current turned off for a minute or so after every three minutes. He has seen no burns by this method but states that care must be taken with more powerful machines. He believes the method should be more widely employed by ophthalmologists, and that it is of especial value in glaucoma secondary to uveitis.

It may be concluded that short-wave therapy is the most efficient method known of producing heat in the deeper tissues and that it is probably of real value in severe infections of the lids and orbit, acute dacryocystitis and probably in certain cases of chronic uveitis. It must be used with care to prevent burns and further observations as to exact dosage are desirable. It would seem to be contra-indicated where the danger of hemorrhage exists.

FEVER THERAPY INDUCED BY LONG- OR SHORT-WAVE DIATHERMY

There can be no doubt that when fever therapy is indicated, it can be very efficiently and constantly obtained by diathermy. The blankets with which long-wave diathermy was employed have rather generally been replaced by cabinets in which a field of short-wave current is generated while the patient is kept dry by the passage of air. The desired temperature can be produced more rapidly in such cabinets than in those employing air-conditioning alone. Even when every precaution is taken to replace lost fluid and salt during the treatments, the method is not without danger. Hence, it should be reserved for serious conditions which do not respond to other forms of therapy, and must not be employed in elderly or infirm patients, especially if increased blood pressure or myocardial lesions are present. It is the routine at Passavant Hospital to require an electrocardiograph in addition to a thorough physical examination before fever therapy in the cabinet is given. The treatment must be supervised throughout by an assistant who is thoroughly familiar with the method and with complicating conditions which may arise during treatment.

Three conditions seem to offer special indications for this form of therapy. The most important of these is gonorrheal ophthalmia in the adult. Pinkerton[12] has reported excellent results in three cases with fever of 107° F. maintained for five to ten hours. Swelling and secretion subsided promptly and smears became negative twenty-four hours after the first treatment. In two cases corneal ulcers were present when treatment was begun and healed promptly. While no other reports have been seen, the success of the method

in other forms of gonorrhea and the severity of the process under former methods of treatment seem to offer a definite indication for the method. Results in the few cases available seem to be definitely superior to those obtained by the usual foreign protein therapy.

The second of the three conditions mentioned is *interstitial keratitis*. The most complete report on the subject is by Culler and Simpson.[13] They employed the Kettering Hypertherm, an air-conditioned cabinet, producing fever of 105° F. which was maintained for five hours. Weekly treatments were given for ten weeks, each treatment being preceded by an injection of bismarsen. They observed eleven cases with twenty affected eyes for eighteen months or longer. At the end of this time no patient had vision of less than 6/30 while vision in eighteen eyes was 6/12 or better. The average period of congestion in the treated cases was two months. Eight of the cases treated had suffered recurrences on other treatment and ten had failed to respond to chemotherapy. It will be noted that the degree and duration of temperature in this series were both considerably less than those used in treating gonococcal infections.

The third condition in which fever therapy seems indicated is optic atrophy in syphilis of the central nervous system. Culler and Simpson report on sixteen cases treated by the same method employed in interstitial keratitis. Central vision remained the same in practically all cases and the fields increased in three. A number of cases have been reported following treatment with malaria by Fischer-Ascher, Heinsius, Gasteiger, Clark and others. In general, results were encouraging to the employment of fever and the advantages of fever produced in the cabinets over malarial inoculations are obvious. Neymann[14] treated seven cases of specific optic atrophy, employing fever of 107.6° for eight hours. Treatments were given twice a week for ten weeks. Three advanced cases went on to blindness. Two showed slight improvement in vision while two remained unchanged. His cases were in general advanced when treatment was begun. When compared with the method of cisternal injections, results in a limited number of cases appear more favorable with the latter method, but it must be considered as more dangerous than fever therapy.

On account of the necessary nursing care and the dangers involved it seems best to reserve high fever therapy for the three conditions just mentioned. Culler and Simpson report good results in luetic iritis and Whitney[15] treated various types of keratitis and iritis with the method, but there seems reason to think that equally

good results could have been obtained in these cases by simpler methods. The use of a lower temperature, 105° F., and a shorter period (four hours) may prove of advantage in certain intractable cases of chronic uveitis, the dangers of such a temperature being negligible when proper precautions are taken.

ROENTGEN THERAPY

This procedure has been mentioned as having proved valuable in the treatment of certain types of keratitis. The rationale of the method, which employs doses so small as to be perfectly safe, is not clear. It is improbable that bacteria are killed although there is some evidence that the herpes virus is especially susceptible to roentgen irradiation. It is more likely, as Case suggests, that leucocytes, which are extremely sensitive to roentgen irradiation, are broken down in the tissues with the liberation of ferments and lytic substances which aid in the body's defensive reaction. Cells involved in the healing of corneal defects may also be stimulated. It is hard to bring scientific proof of such phenomena. Clinical experience, however, has convinced me that in dendritic ulcer, superficial punctate keratitis, degenerative ulcers or old scars, and a few other types of keratitis, one or two small roentgen exposures may be of great value. Such treatments have been given for me by Dr. James Case in ten cases, chiefly of dendritic keratitis, most of which had resisted other treatment, or had recurred after such treatment. Most of these ulcers healed within one or two days of the first treatment and a second treatment was seldom required. In no case has any evidence of damage to the lens been seen which is what could be expected from such small doses. Such treatments are given, of course, without any form of protective shell; 80 to 90 roentgen units of unfiltered radition with current of 100 Kv. was given at each treatment.

Roentgentherapy is of great value in severe cases of blepharitis. Three doses of 80-90 roentgen units each are given at intervals of two weeks, a mercury-filled glass or a lead shell being placed beneath the lids to protect the cornea and lens. Cases of many years' standing which have resisted the usual forms of treatment have practically always responded to this treatment. The use of a mild ointment, usually of 2 per cent zinc oxide in hydrated lanolin, is used for some time to dissolve crusts and prevent recurrences. In a few cases a second course of treatment after a year or more has been necessary. Such dosage has never caused loss of lashes in our cases and no damage to the lens has been observed.

In a few cases of early post-operative and post-traumatic infection, results have been encouraging enough to deserve mention, although the number of cases is too small to justify definite conclusions.

SUMMARY

1. Phototherapy is probably of value in herpetic and a few other forms of superficial keratitis.

2. The infra-red lamp is a useful and practical instrument for applying ordinary heat.

3. Local short-wave diathermy may prove to be of considerable clinical value in the treatment of chronic uveitis and in cellulitis of the lids and orbit. Standardization of dosage is desirable.

4. General fever therapy by the use of diathermy cabinets is of value in gonorrheal ophthalmia, interstitial keratitis and specific optic atrophy.

5. Roentgentherapy is of undoubted value in obstinate cases of blepharitis, dendritic keratitis, superficial punctate keratitis in its more severe forms and possibly in disciform keratitis.

BIBLIOGRAPHY

1. Gifford, Sanford R.: Trans. Am. Acad. of Oph. and Oto. 1928:33, p. 195.
" " Handbook of Ocular Therapeutics. 1937, p. 103.
2. Nugent: Arch. Phys. Therapy. 1936:17, p. 234.
3. Hoffman and Kunz: Arch. f. Ophth. 1934:132, p. 155.
4. Kokott: Klin. Monatsbl. f. Augenh. 1936:97, p. 448.
5. Gutsch: Deut. Oph. Ges. 1936:51, p. 221.
6. Scheyling: Klin. Monatsbl. f. Augenh. 1937:98, p. 387.
7. Carlotti: Klin. Monatsbl. f. Augenh. 1937:98, p. 575.
8. Kuwahara: Klin. Monatsbl. f. Augenh. 1937:98, p. 576.
9. Krause: Ztschr. f. Augenh. 1936:89, p. 266.
10. Kiewe: Klin. Monatsbl. f. Augenh. 1935:95, p. 108.
11. Walker: Trans. Am. Ophth. Soc. 1935:71.
12. Pinkerton: Am. J. Ophth. 1937:20, p. 63.
13. Culler and Simpson: Arch. of Ophth. 1936:15, p. 624.
14. Neymann: *Artificial Fever Therapy*, Thomas, Springfield, Ill., 1937.
15. Whitney: J. A. M. A. 1935:104, p. 1794.

Symposium on Physical Therapy

* * *

USE OF PHYSICAL THERAPY IN OTOLARYNGOLOGY

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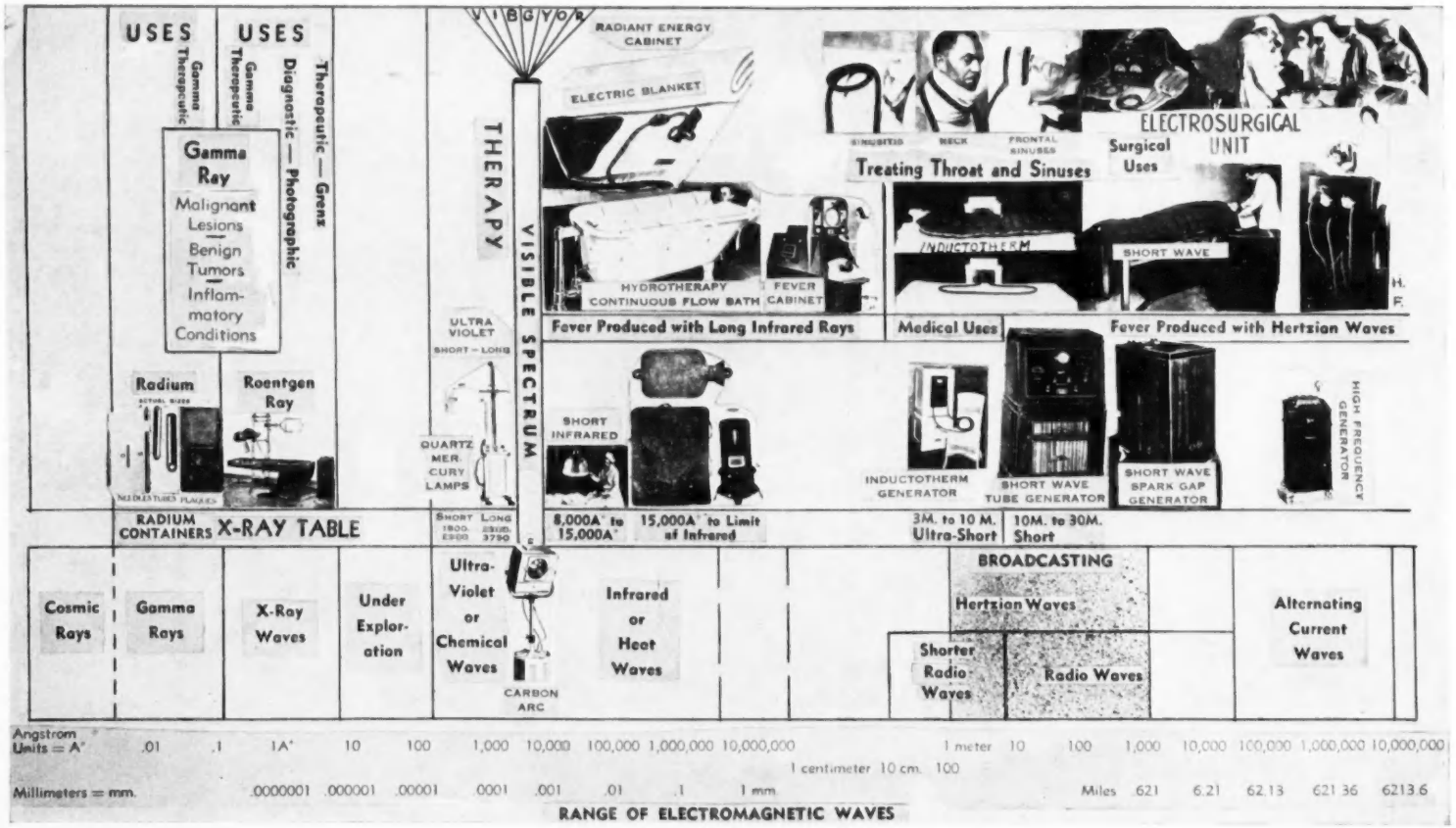
PREFACE — The following picture (see next page), I had made because:

1. It helps each one to visualize the *Universe of Energy* in which he is living.
2. It enables each one to orient himself in relation to various fields of energy found in the Spectrum of Radiation.
3. It reveals the genius of physicists in their adventure to solve mysteries of the universe.
4. It shows the work of inventors who created generators which produce energy found in this spectrum.
5. It demonstrates what medical men have done and are doing in applying this energy therapeutically.

In 1666, Sir Isaac Newton passed a ray of sunlight through a prism and discovered it was composed of seven colors — violet, indigo, blue, green, yellow, orange and red. Further research revealed these colors were due to energy coming from the sun in an undulating, wave-like motion. Finally, investigators were able to measure the distance from the crest of one wave to another. On the violet side it measured 3750 A° (Angstrom units, the unit being 1/10,000,000 of a millimeter, and was named after Angstrom, a Swedish physicist); and on the red side, 7500 A°. This is known as the Visible or Solar Spectrum.

Continued research revealed invisible energy on either side of the Visible Spectrum. On the violet side the wave length became shorter and shorter, and the various fields were named ultraviolet, x-ray, gamma ray, and cosmic ray. On the red side the wave length



RANGE OF ELECTROMAGNETIC WAVES

was longer and longer and was named infra-red and Hertzian waves (ultra-short, short, and long radio waves and the high frequency). The name applied to this field of energy from extreme limits of the cosmic and high frequency fields is the Spectrum of Radiation or Electromagnetic Wave Spectrum.

Following the discoveries of physicists, inventors created generators which produce energy found in these fields as you will observe by studying the picture. Medical researchers then applied this energy therapeutically and they are finding it of service in treating various diseases.

THREE SECTIONS OF THE SPECTRUM

For convenience in studying the therapeutic application of this energy, let us make three groups of the Spectrum of Radiation:

1. Radium and Roentgen Rays — these two forces have deep penetrating qualities and are destructive to certain cells and tissues.
2. Light rays, ultraviolet, solar spectrum and infra-red — the action of these rays is actinic and thermal, but they have little penetrating power.
3. Hertzian waves, the ultra-short, the short and long radio waves and the high frequency current. The energy of Hertzian waves is thermal in action on the body and is used both medically and surgically.

GAMMA RAY A PRODUCT OF RADIUM AND ROENTGEN RAY

The gamma ray, a product of radium, is essentially a therapeutic agent. Though it has deep penetrating power, it is used especially where it can be applied locally as:

1. On superficial lesions in the skin, a plaque is used.
2. In cavities, as in the nose, sinuses and lungs, a tube is placed within so as to irradiate the diseased portion.
3. In tumors, as of tongue, tonsils, naso-pharynx, etc., needles or radon seeds are implanted in the lesion.

The roentgen ray is both a therapeutic and diagnostic measure. Every otolaryngologist is so familiar with it as a diagnostic agent that there is no need of comment. Often the roentgen ray is used therapeutically where radium could be applied. The advantage in

using it instead of radium is where the lesion is deep within the tissue or where large areas demand irradiation. Since the shortest roentgen ray is a gamma ray, the question is not which is the best therapeutic agent, but which can be used most advantageously.

COUTARD'S TECHNIQUE

Since Coutard has been active in quickening the interest of medical men as to the therapeutic value of the roentgen ray in malignant tumors, and since he is referred to so frequently, it is well here to list the dominant principles of his technique as outlined by Cutler:[1]

1. A correct and adequate diagnosis
2. Low intensity of radiation
3. Continuity of treatment
4. Optimum total time of irradiation confined within the safe limits of radiation immunity.

Following the technique of Coutard, Cutler says, "The patients are treated twice daily, consecutively without interruption until the cycle is completed," i.e., from twenty to sixty days.

RADIOSENSITIVITY

Roentgenologic researchers have discovered that when a body is exposed to roentgen rays, various cells are affected differently — "Some are destroyed, some injured, but regenerate later, and some do not show any deleterious effect." The reactions of cells to irradiation determine their sensitivity to the rays.

Beginning with the most sensitive cells, i.e., those first to evidence action of the rays, the following table shows, in the sequential order of destruction, the effect of irradiation.

1. Lymphoid cells
2. Polymorphonuclear leucocytes and Eosinophils
3. Epithelial cells
4. Endothelial cells
5. Connective tissue cells
6. Muscle cells
7. Bone cells
8. Fat cells
9. Nerve cells

Further, it has "been shown conclusively that the younger and the more active the cell, from a metabolic point of view, the more susceptible it is to the influence of rays."[2]

The successful use of these rays therapeutically is based on the work of pathologists whose reports show the histopathological structure of the tumor and its classification and, therefore, inform the surgeon and the radiologist whether the lesion is sensitive or resistant to radiation. We, as otolaryngologists, are interested in the following classification of tumors relative to their radiosensitivity:

Radiosensitive Tumors	1. Lymphoblastoma
	2. Lympho-Epithelioma
	3. Basal Cell Epithelioma
	4. Transitional Cell Epithelioma
	5. Hemangioma
	6. Hemangio-Endothelioma
Moderately Radiosensitive Tumors	1. Epithelioma of tonsils
	2. Epithelioma of bronchus
	3. Epithelioma of lip
	4. Epithelioma of mouth and tongue
	5. Epithelioma (squamous cell) of skin
Radioresistant Tumors	1. Fibrosarcoma
	2. Carcinoma of Esophagus
	3. Adenocarcinoma
	4. Mixed Tumor of Parotid Gland
	5. Sarcoma of bone
	6. Melanoblastoma
	7. True teratoma
	8. Neurofibroma
	9. Fibroma
	10. Chondroma
	11. Lipoma
	12. Myxoma
	13. Myxosarcoma

Today we find an ever-increasing interest in the therapeutic use of radium and roentgen rays. In this city, at a recent meeting of the "Fifth International Congress of Radiology," almost a score of papers were read on subjects of interest to O. R. L. men.

NEED OF INFORMATION

We know that many Academy members affiliated with large clinics are familiar with what is being done with these agents. The larger number, however, is not so fortunate and, because the press is publishing so many articles on the use of these modalities as a method of curing diseases in which we specialize, it is imperative that every Academy member be informed—hence, this symposium.

Considering our present knowledge and experience, we are convinced that surgery holds first place in cancer therapy. It has taken many years to attain its present high degree of efficiency and, established scientifically as it is, there need be no fear but that surgery will ever have a place in the treatment of diseases. Let us here emphasize the fact that we are not presenting a brief advocating the use of radium and roentgen ray to the exclusion of surgery. Though in the last decade progress has been made in using radium and roentgen ray therapeutically, it will take many years of intensive research work to determine and establish the full therapeutic value of these agents. Through the combined use of radiology and surgery, progress is being made in cancer therapy. For years I have felt that radiologists have over-rated and made exaggerated statements as to the value of these physical forces in treating diseases, but as I searched the literature and found evidence of their worth, not only in malignant tumors, but also on other pathological conditions, even if reports are only partly true, I have wished every otolaryngologist might have a more perfect knowledge of the work that is being done by radiologists.

MODERN CANCER THERAPY

As one studies the subject, the fact becomes more patent that to succeed in cancer therapy, there must be a closer cooperation between medical men. General Frank Hines,[3] an Administrator of a Veteran's Tumor Clinic, says of these clinics, "Each tumor clinic is so organized that every patient is given the benefit of consultation by a group of experts and consultants, inasmuch as no single physician is competent to treat cancer, but the combined efforts of the surgeon, the radiotherapist, the pathologist, physicist and other specialists are needed to decide upon the diagnosis and treatment in each case."

Often lesions are found where more than one therapeutic measure should be used, e.g.:

1. Some tumors respond favorably to either radium or roentgen ray therapy.
2. In others, surgery, followed with irradiation by either radium or roentgen ray or both may be indicated.
3. Still in others, coagulation followed by using either of these or both may be the optimum therapeutic measure.

In evaluating the worth of these agents in treating carcinoma, Cutler,[4] in his article on "Radiosensitivity," says, "The outstanding successes in radiation therapy have been accomplished in carcinoma of the skin, lip, tongue, oral mucous membrane, tonsils, pharynx, larynx, etc. It has been noticed that it is precisely in these localities that irradiation has largely replaced surgical procedures."

New and Cabot[5] in a paper on the "Treatment of Malignant Tumors of the Upper Jaw and Antrum" say:

1. "Malignant tumors of the upper jaw and antrum are curable.
2. "Surgical diathermy and irradiation are the most efficient measures of treatment.
3. "Biopsies should be made in all cases at the time of operation as an aid in determining treatment."

BENIGN TUMORS

According to Robinson[6] the following benign tumors taken from a long list of others respond favorably to radium therapy:

1. Hemangioma
2. Polypoid ethmoiditis
3. Fibroma of Pharynx
4. Rhinoscleroma
5. Tubercular adenitis

In hemangioma the action of the gamma rays on the blood vessels produce "a proliferation, vacuolation and degeneration of the endothelial cells with leucocytic infiltration of the walls and subsequent destruction of their caliber." Even in large hemangiomas where surgery is indicated, it is well to irradiate first.

In polypoid ethmoiditis, "in a series of more than 100 cases, it has been found that in a high percentage of cases, chronic nasal discharge, headache and asthmatic attacks have been relieved." Here, surgery should always precede irradiation.

"Irradiation of fibromas with radium before operating causes the neoplasm to shrink and reduces danger of hemorrhage."

Early rhinoscleroma cases are cured and advanced cases relieved by radiation, "reducing the loose granulation fibrillar meshwork in the submucosa to a dense fibrous tissue."

Either radium or roentgen rays, in cervical adenitis, "produces an inflammatory reaction in the tissues, causing a shrinkage and gradual disappearance of the lymph nodes and a healing of the discharging sinuses." Cures are obtained in a majority of cases.

INFLAMMATORY LESIONS

One finds many articles on the treatment of inflammatory lesions by irradiation. Desjardins[7] says, "This method of treatment is not used as widely as it might be, probably because its value is not generally realized." In the treatment of 1018 consecutive cases of superficial inflammatory lesions, Friedman[8] lists:

1. Lymph adenitis, acute and chronic..... 101
2. Cellulitis 354
3. Carbuncle 206
4. Furunculosis 114
5. Erysipelas 39
6. Mastoiditis 21

Etc.

His summation states that radiation therapy in superficial infections offers a method of treatment that is highly beneficial, curative in early cases, and shortening the clinical course in the more advanced processes.

Before leaving this part of my subject I must refer you to the following articles on irradiation:

1. Bronchiectasis by Berck and Harris[9]
2. Erysipelas by Hodges and Berger[10]
3. Mastoiditis by Schillinger[11]
4. Sinusitis by Butler and Woolley[12]

Again I would remind you that I am giving what is reported by radiologists as being accomplished through irradiation. While surgery still holds first place and will ever be needed, we must have an open mind and always accept facts in our search for the best in therapy.

PHOTOTHERAPY

In attempting to evaluate the therapeutic uses of light, one soon becomes bewildered. Though it is known that light has been used for centuries in curing disease, there is little evidence that it has been used scientifically. In recent years many volumes and myriads of articles have been published by researchers, empiricists and manufacturers of generators which produce light rays; many of these articles cause confusion because of contradictory statements. Many of them have extolled the use of light as a preventive and cure of diseases. So thorough has been the propaganda that today the laity is frantically seeking methods to secure a bronzed skin by natural sunlight or light rays produced by man-made generators. Multitudes have been led to believe that in being tanned they are being immunized against infection. In the end, they have learned that light is no therapeutic panacea, and that they have been deceived and deluded. The research workers on light, thus far, while progress has been made, have failed in proving the therapeutic value of the various wave lengths of energy found in this portion of the Spectrum of Radiation.

PHYSIOLOGICAL ACTION OF LIGHT RAYS

In using generators which produce these various rays, one should remember:

1. The action of all these rays is superficial.
2. The action of ultraviolet rays is actinic, bactericidal and biological.
3. The action of the visible rays is actinic and thermal.
4. The action of the infra-red rays is thermal.
5. The action of all is erythemogenic.
6. Ultraviolet produces erythema three to six hours after exposure and it lasts long.
7. Visible rays produce both quick and short lasting, and delayed and long lasting erythema.
8. Infra-red produces quick erythema and short lasting.
9. The least penetrating rays are the short ultraviolet.
10. The deepest penetrating rays are the short infra-red.

RAYS OF DIFFERENT WAVE LENGTHS WORK TOGETHER

Laurens[13] says: "The use of radiant energy (natural or artificial sunshine) for therapeutic or scientific purposes must be built on a strong foundation of physics, and users of lamps, since they are essentially trying to imitate sunlight and its effects, must know something about the physics of their sunlight. There is a general, almost universal, but wrong tendency to limit the biological effects of radiant energy and their therapeutic action to the ultraviolet and particularly to the region between 2900 and 3130 angstroms. That radiation is solely or mainly active because the ultraviolet fraction has been unequivocally demonstrated in only a few instances (rickets and infantile tetany). There is considerable evidence on the other hand that the luminous and infra-red rays, even if not specific in their action, aid the ultraviolet rays."

SUNLIGHT NOT THE ONLY VITALIZING FACTOR

That sunlight is invigorating is common knowledge. But, sunlight is only one of many vitalizing factors necessary to prevent disease and to maintain health. A balanced diet with correct and proportioned supply of vitamins, hygienic surroundings, proper exercise, a sufficient amount of fluids, pure air, normally functioning endocrines, drugs as indicated, all of these are essential. Light may be synergistic to any or to all of these.

Coblentz[14] says: "Except for the question of intensity and wave length, physically there is no marked distinction between the various regions of the spectrum called 'ultraviolet', 'visible', and 'infra-red'." Chemically and physiologically there is a difference in their action. The short ultraviolet rays are bactericidal in action, the long ultraviolet rays are biological, and the infra-red are thermal. All are superficial in action and therapeutically their use in Ear, Nose and Throat work has been exaggerated and to date has been disappointing.

Under "Ultraviolet Therapy," in the index of the Handbook on Physical Therapy, put out by the Council, over two score diseases are listed as amenable to treatment, but in the text, MacKee[15] says: "In all probability the bacteriologic effect of the easily absorbed shorter waves is negligible. The possible influence of the more penetrating visible light rays and infra-red rays has not been carefully studied in their therapeutic relation to the near and far ultraviolet rays."

In his book on "Light Therapy" Krusen[16] says, "Even with our present rather limited and chaotic understanding of light therapy,

it may be said to be distinctly valuable in a fairly large number of pathological conditions, and to offer great promise of still further usefulness when more accurate methods of administration are developed, and when we have attained more specific knowledge of therapeutic action of various wave lengths."

ULTRAVIOLET THERAPY

Our present knowledge leads us to believe there are three conditions in which ultraviolet therapy has almost specific action — rickets, infantile tetany and spasmophilia. In the following conditions, ultraviolet therapy may be of service to O.R.L. workers. Aural tuberculosis;[17] lupus of nasal mucosa;[18] laryngitis (non-tubercular;[19] nasal polypi, following removal;[18] eczema;[15] erysipelas;[20] tuberculosis of the larynx;[21] in pruritis of aural canal, also in aspergillus infection, I have found it beneficial. In Vincent's infection, while it is good, experience has led me to depend on neoarsphenamine, intravenously administered, and on other drugs locally applied. In trachæitis a second degree erythema on the chest has proved helpful. The field where ultraviolet therapy seems to be most helpful is in extra-pulmonary tubercular conditions.

When applied systemically, the combined action of ultraviolet, visible spectrum and infra-red may be of service in healing sluggish, indolent wounds; namely, in postoperative mastoidectomies, also in secondary anæmia, tubercular lymph nodes (with other therapy), tuberculosis of larynx, and aural tuberculosis.

INFRA-RED THERAPY

Since the action of the infra-red ray is thermal and its use produces hyperemia, analgesia, and is antispasmodic, germicidal, absorptive and nutritive, it is obviously of service when used locally in rhinitis, sinusitis (after drainage), laryngitis, otitis media (after myringotomy), mastoiditis, furunculosis, myalgia, and neuritis. In these conditions the short ray, 8,000-15,000 A, because of its penetration, may be used and for forty-five minutes or longer at each treatment. The treatment should be repeated as often as is necessary to effect normal physiological action. Diathermy, short wave or conventional (HF) also may be used in these conditions.

HERTZIAN WAVES

Energy, in the Hertzian wave field is used both medically and surgically. The therapeutic value of this agent is heat, produced by high frequency generators of which we shall mention three. (1)

In conventional diathermy (oscillations from one million to two million per second) the currents are applied with contact metal electrodes. (2) In short wave diathermy (oscillations from ten million to one hundred million per second) the electrodes are metallic plates insulated by rubber and between the skin and electrode a piece of felt or towel is placed to prevent accumulating surface moisture into small conducting areas, which might produce burns. (3) In electromagnetic induction diathermy, the current is conducted to the patient through a flexible, well insulated cable which is wound around the part to be treated as in using electrodes in short wave diathermy. In conventional diathermy, heat is produced by resistance of current. In short wave diathermy and in inductothermy it is not positively known how the heat is produced, perhaps through molecular action or eddy currents.

HEAT — THE ONLY THERAPEUTIC AGENT

Though we have these three methods of applying electric currents, at the present time it is believed the only therapeutic effect is by the heat produced. The changes due to heat from diathermy are:

1. Capillaries are opened
2. Blood flow is increased
3. Tissue metabolism is accelerated
4. Rate of exchange between blood and tissue is augmented.
5. Oxygen carried into veins may be increased 33%
6. Activity of leucocytes in phagocytosis
7. Increased metabolism

Though claims have been made that short wave diathermy is useful in furuncles and abscesses, a member of the Council[22] states that, "It is questionable whether the greater heat of short wave diathermy applied for short periods could replace hot moist dressings or improve on the results obtained from them." However, Bierman and Schwartzschild[22] say, "It is our definite impression that when drainage does exist, the infected process usually appears to heal very much more rapidly following short wave application than with any other forms of treatment we have used heretofore." This last statement is in keeping with the physiological action of heat in accelerating the blood supply in the pathological area, therefore, diathermy is obviously indicated in sinusitis, mastoiditis and otitis media after drainage has been established.

MASTOID INFECTION

In a patient, often more than one modality may be of service. Short infra-red for moderately deep heating, diathermy for deep heating and, where there is bone necrosis, ultraviolet radiation for calcium fixation are of service.

When clinical, laboratory and roentgen ray findings indicate operative procedure, after treating as indicated herein, one finds the abscess well walled off, and postoperative recovery is more rapid. From literature I find myself at variance with eminent men relative to the use of diathermy in mastoiditis, and I am made to wonder if I am in error or if we differ in technique. At times, I have taken the place of my technician in order to watch the results during the treatment, and have observed that at the beginning there would be a tenacious, ropy, purulent discharge which gradually changed during the forty-five minutes of treatment to a serosanguinous character, brought about by producing heat in the petrous and mastoid bones. Using heat therapeutically, whether infra-red or diathermy, has convinced me that it should be repeated (p.r.n.) as a drug is used to maintain that action, and that often an operation is prevented in mastoiditis.

SURGICAL DIATHERMY

For surgical diathermy we have three modalities:

1. Fulguration or desiccation, which means drying. With this method the current is usually used from only one outlet of the generator.
2. Coagulation is a cooking of tissues and is produced by using two outlets of the generator. With coagulation the tissue may be destroyed many millimeters in all directions.
3. Cutting electrically is produced by using a large pad on which the patient lies, connected to one outlet of the generator and connected with the other outlet is an insulated handle in which a fine wire or blade is fastened, which serves as a scalpel. By the current being conducted to and concentrated on the wire or blade, an incision can be made when applied to tissues, as with a sharp instrument.

The advantage in using these modalities is that they produce an instantaneous occlusion of lymphatic vessels and, therefore, prevent metastasis.

The primary indication for using these agents is in malignancies, for reasons just given.

Fulguration, as a rule, should be used only on superficial and benign lesions as (1) small hematoma, (2) small nasal polypi, etc.; Howard[23] recommends its use for contact ulcers of the vocal cords.

Coagulation is of service when one desires deep destruction of tissue. Care should ever be taken not to injure large blood vessels lest there be a severe delayed hemorrhage due to sloughing. This can be used in both benign and malignant lesions. It is of service in synechia in the nose, hypertrophied turbinates, etc., also in lesions in the mouth, sinuses, pharynx and larynx.

Cutting electrically is indicated in malignancies as of the face, lips, tongue, oral cavities and sinuses (where also coagulation is used).

ELECTRO-COAGULATION OF TONSILS

Some eminent men and many lesser lights advocate one or more of these modalities in removing tonsils. Personally, I have never seen a case where any one of these modalities was indicated in tonsil work. I believe its advocacy is proving to be injurious to the public, because many men, who are not prepared to do surgery, are easily convinced that with an outfit they can remove tonsils, about which operation they know little or nothing. The end result is that the crypts are sealed with scar tissue or buried by an overgrowth of mucous membrane, and the infection is thrown back into the system. A far better method of treating tonsils is the use of radium or roentgen ray and this in my hands has proved to be a disappointment.

OTHER USES OF THESE FORCES

Hippocrates is alleged to have said that if he could produce fever, he could cure many diseases. Today artificial fever is produced by applying heat externally and internally to the human body. Some modalities used for the first are the hot water bath, radiant cabinet, electric blanket, and the hyperpyrexator; some used for the second are the inductotherm, the short wave and the conventional diathermy. While much research is being done in fever therapy, little has been done in ear, nose and throat work.

On the use of artificial fever in sensitizing malignant tumors, Warren[24] says, "It will probably take another ten years before we have much factual data about the effects of fever and radiation of malignant tissue in human cases. It is very difficult to unravel the exact dosage relationships between the fever and fractional

X-ray therapy, but I still believe that the suggestions made in the paper are valid, that the combination of radiation therapy and fever is more destructive than radiation therapy alone."

Some men, among whom are Fuchs,[25] believe malignant lesions are made more sensitive to irradiation through heating with diathermy. Hollander[26] says, "Our results were exactly the same with or without the use of diathermy — 'In far advanced cases, however, I am using short wave diathermy for it does give a certain amount of alleviation of pain, which I believe is the most important thing.'"

Iontophoresis for hay fever has its proponents. While it may have a place in nasal therapy, it should be used only in selected cases.

Our study of the Spectrum of Radiation has shown us that:

1. Progress is being made in the therapeutic use of radium and the roentgen ray.
2. The action of light, ultraviolet, infra-red and luminous rays has not been scientifically established though evidence enough has been given to show that through further research it may prove to be a potent therapeutic agent.
3. The medical and surgical use of heat produced by the infra-red rays and the Hertzian waves is of value in combating disease.
4. Much more research is necessary before the full therapeutic value of energy in the Spectrum is known.
5. There must be closer cooperation between and group work among physicians in order to secure optimum therapeutic results.
6. We must "try all things, prove all things, hold fast to that which is good."

REFERENCES

1. Cutler, Max: *"Radium Dosage and Technique in Carcinoma of the Tonsil, Pharynx and Larynx."* Am. J. of Roentgenol. 33:690, (May) 1935.
2. Desjardins, A. U.: *"Radiotherapy."* Handbook of Physical Therapy, Chicago, A.M.A. Press, 1936, p. 262.
3. Hines, General Frank T.: *"Government Surgeons Mobilized to Combat Increase of Disease Among Ex-Soldiers."* Science News Letter, Sept. 4, 1937.
4. Cutler, Max: *"The Problem of Radiosensitivity."* J.A.M.A. 103:1204, (Oct. 20) 1934.

5. New, Gordon B., and Cabot, Clyde M.: *"The Curability of Malignant Tumors of the Upper Jaw and Antrum."* Surg. Gynec. and Obst. 60: 971, (May) 1935.
6. Robinson, G. A.: *"Benign Lesions of the Eye, Ear, Nose and Throat."* Am. J. Roentgenol. 33:801, (June) 1935.
7. Desjardins, A. U.: *"Radiotherapy for Inflammatory Conditions."* J.A. M.A. 96:401, (Feb. 7) 1931.
8. Friedman, A. B.: *"Superficial Inflammatory Diseases."* Am. J. of Surg. 25:107, (July) 1934.
9. Berck, Maurice, and Harris, William: *"Roentgen Therapy for Bronchiectasis."* J.A.M.A. 108:517, (Feb. 13) 1937.
10. Hodges, F. M., and Berger, R. A.: *"Roentgen Therapy of Some Infections."* J.A.M.A. 107:1551, (Nov. 7) 1936.
11. Schillinger, Rapheal: *"Roentgen Therapy in Acute Mastoiditis."* Paper read at Congress of Physical Therapy, Cincinnati, Sept., 1936. Personal communication.
12. Butler, F. E., and Wooley, L. M.: *"Roentgen Therapy in Chronic Sinusitis."* Radiology. 23:528, (Nov.) 1934.
13. Laurens, Henry: *"Factors Influencing Choice of Source of Radiant Energy (Ultraviolet, Luminous and Infra-Red)."* J.A.M.A. 103:1447, (Nov. 10) 1934.
14. Coblentz, W. W.: *"Sources of Ultraviolet and Infra-red Radiation Used in Therapy."* Handbook of Physical Therapy, Chicago, A.M.A. Press, 1936, p. 170.
15. MacKee, George M. *"Ultraviolet Therapy in Dermatology."* Handbook of Physical Therapy, Chicago, A.M.A. Press, 1936, p. 236.
16. Krusen, Frank Hammond: *"Light Therapy."* N. Y., Hoeber, 1933, p. 163.
17. Mayer, E.: *"Present Status of Light Therapy."* J.A.M.A. 98:221, (Jan. 16) 1932.
18. Deck, E. J.: *"Treatment of Nasal Infections by the Super-Kromayer Lamp."* Brit. J. Phys. Med. 7:6, (April) 1932.
19. Humphris, F. H.: *"Artificial Sunlight and Its Therapeutic Uses."* Ed. 5, Oxford Univ. Press, 1927, p. 4.
20. Nightingale, Louis M., and Starr, Saul: *"The Treatment of Erysipelas in Children."* J.A.M.A. 102:761, (Mar. 10) 1934.
21. Mayer, E.: Internat. Clin. 2:203, (June) 1932.
22. Coulter, John S.: *"Medical Diathermy."* Handbook of Physical Therapy, Chicago, A.M.A. Press, 1936, p. 344.
23. Howard, R. E.: *"Fulguration of Contact Ulcer of the Vocal Cords."* Paper read at Congress of Physical Therapy, Cincinnati, Sept., 1937. Personal communication.
24. Warren, Stafford B.: *"Preliminary Studies of Effect of Artificial Fever upon Hopeless Tumor Cases."* Am. J. of Roentg. 33:75, (Jan.) 1935. Personal communication.
25. Fuchs: *"Sensitization of Neoplasms by Means of Short Waves."* J.A. M.A. 106:2115, (June 13) 1936.
26. Hollander, Lester (Pittsburgh, Pa.): Personal communication.

Section on Ophthalmology

The American Academy
of Ophthalmology and Otolaryngology
Forty-Second Annual Meeting
Chicago 1937

CHLOROMA: REPORT OF A CASE WITH HEMATOLOGIC STUDY

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SINCE exophthalmos is usually the first symptom of chloroma, a discussion of these cases deserves a place in ophthalmic literature, even though the subject remains primarily in the realm of the hematologist. A complete review of the literature would be superfluous at this time, since this has been done so recently by Gump, Hester and Lohr.[1]

The gross anatomic features of chloroma were first described by Burns[2] in 1823, who noted the green orbital tumors and fatal outcome. King[3] was the first to use the name chloroma in 1853. Von Recklinghausen[4] in 1885 mentioned that the histologic pictures of chloroma and leukemia were similar but made no correlating blood studies. In 1893, Dock[5] discussed the relationship between chloroma and leukemia and in 1904 he and Warthin[6] definitely established this relationship. In 1910, Lehndorff[7] reviewed the seventy-three cases which had been reported and considered fifty-six of these to be of lymphoid origin. Inferior hematologic methods and the generally accepted lymphoid nature of chloroma were probably responsible for the slow recognition of the myeloid nature of the disease. At the present time, most hematologists agree that the process is myelogenous.

In many of the cases that have been reported as chloroma, the diagnosis may be considered doubtful. In several instances there was neither biopsy nor autopsy, and therefore the green color and other characteristics of the tumor were not disclosed. This is particularly true of the cases reported by Gould and LeWald[8] in which the separation of the suture lines of the skull was stressed as an important diagnostic sign. Although this is evident in some cases, it is a rare finding. Their diagnosis may be questioned also because the blood examinations did not show leukemia, the biopsy of the lymph glands did not show the green color, and autopsy was

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not performed. True aleukemic forms of the disease are rarely encountered, but relatively aleukemic stages may be present.

Brannan's[9] review in 1926 left little doubt of the myelogenous nature of the process and suggested that many cases previously reported as chloroma were of doubtful authenticity. Nevertheless, of seven cases reported by Feer[10] as late as 1927, three were classed as lymphoid, but one of these was diagnosed as lymphosarcoma at autopsy. His second case showed leukopenia and nearly 90 per cent lymphocytes. In none of these cases was oxidase or supravital staining technic used. Even the oxidase reaction does not differentiate lymphocytes from myeloblasts, and it is easily seen that immature myeloblasts may have been mistaken for lymphocytes.

As yet there have been no cases of definitely proved chloroma in which the outcome was not rapidly fatal. Washburn[11] in 1930 reported a case with cure by roentgen therapy two and one-half years after appearance of the tumor. This patient had a definite but moderate leukemia with an abnormal number of immature cells of the myeloid type (myelocytes) ranging from 5 to 26 per cent. A green tumor of the frontal region was found at biopsy. According to a recent communication from the author, the child is still living. Distinguished pathologists in various parts of the country recently have examined sections of tissue from the tumor, but they have failed to agree on the diagnosis. Some have called it xanthoma, some chloroma, and others have been unwilling to classify it. Washburn says "My own feeling is that in referring to the case it should be mentioned that the diagnosis of chloroma is open to question in spite of the fact that I still believe it is the most plausible diagnosis." This is the only case in which roentgen irradiation was administered in a relatively long course of small doses rather than in full erythema doses. It may be significant, also, that the treatment covered practically the entire body and was not confined to the visible manifestations of the tumor. In twenty-four treatments over a period of six months, the entire body was irradiated, except for the hands and feet. Washburn was unable to find in the literature that similar treatment had been used in any other case of this type, nor was I. At the conclusion of the treatments, the blood count was normal and no immature forms were evident.

Several others, including King[12] and Merculov,[13] have reported apparent temporary benefit from local irradiation. On the other hand, Gamble[14] and other observers have reported rapid progression of the disease in spite of roentgen treatment.

In many of the articles on chloroma, too much stress has been laid on so-called characteristic findings such as separation of sutures, involvement of the bones of the skull, papilledema, subconjunctival hemorrhages, which really represent individual manifestations which may or may not be present. This has led to considerable confusion, in that the absence of these conditions may tend to lead the observer away from the diagnosis. Why make it hard? The subjective and objective signs and symptoms are dependent merely on the situation of the tumor, which may be any place in the body.

It is necessary, then, that a clear definition of chloroma be made, if possible. Localized growths of myeloid elements anywhere in the body, as demonstrated by biopsy or autopsy, and myelogenous leukemia progressing rapidly to a fatal termination are the outstanding features of the disease. It appears more often in children. When adults are affected, the condition seems to occur more frequently after the fourth decade. The underlying pathologic change consists of a primary hyperplasia of bone marrow which may produce masses of immature white blood cells beneath or in the periosteum of the flat bones with metastasis through the blood stream to distant tissues. The hyperplastic areas in the bone marrow increase in size and the bony trabeculæ become rarefied. If they break through they produce flattened greenish tumors over the inner surface of the skull, the vertebræ, and the ribs. The diagnosis can not be made by roentgenographic or blood studies alone. The green color should be present if the term chloroma is to be employed. However, Gump et al.[1] state that the tumors may not be green.

Thus far the cause of the green color of the tumors has not definitely been determined. It is assumed that it is bound up in some way with the metaplasia of the myeloid cell which is peculiar to this type of leukemia, but this color is not necessarily characteristic of the color of bone marrow in other types of leukemia. Boots[15] showed that the color faded rapidly on exposure to light and air and that formalin specimens became green again in hydrogen peroxide. He also found the green color in the buffy coat of the white cells in a case of myelogenous leukemia, but not in the cells from a patient with lymphatic leukemia whose white count was equally high.

Chloroma is definitely a leukemic disease and may be considered as an unusual form of myelogenous leukemia. In this sense it is a distinct entity. Its neoplastic properties can not be questioned,

and chloroma in myelogenous leukemia may be a counterpart of lymphosarcoma seen in lymphatic leukemia.

Case Report — A white boy, aged four years, was examined on November 23, 1935, because of a swelling in the left upper lid. This had appeared about two months before and had increased in size, gradually at first, but for the last two weeks had displayed rapid growth. The child felt perfectly well and had no other complaints. Except for whooping cough a year before the child had had no illnesses and his birth and development had been normal in every way.

Physical examination revealed that the boy was well nourished and of normal development. He weighed 32 pounds. Except for a

FIG. 1 — (Frost) — Photographs of patient showing supra-orbital tumor and exophthalmos. (December 1, 1935)

few palpable posterior cervical and axillary lymph nodes, exophthalmos and downward displacement of the left eye were the only abnormal findings. The exophthalmos was caused by a circumscribed hard tumor mass slightly visible, somewhat nodular, between the globe and the lateral portion of the roof of the orbit. It apparently extended far back in the orbit and was somewhat adherent to the periosteum, causing proptosis of 8 mm. and downward displacement. There was no edema of the conjunctiva, the cornea was clear, the pupil was round and equal to that of the right eye, and normally active. No lagophthalmos, loss of motility or di-

plopia could be demonstrated. The vision was normal, as far as could be determined by illiterate tests, and the fundus showed normal findings (Fig. 1).

The family physician reported that examination of the urine and blood had shown nothing abnormal. A roentgenogram of the skull and orbit revealed no bone change or destruction.

The patient was admitted to the University Hospital on December 2, 1935. In the interval since examination there had been a noticeable enlargement of the tumor of the left orbit, and in addition another tumor had appeared in the same position in the right orbit. This was not detected until the child was anesthetized for operation. The blood count at this time showed 32,000 white cells, with approximately 82 per cent myelocytes.

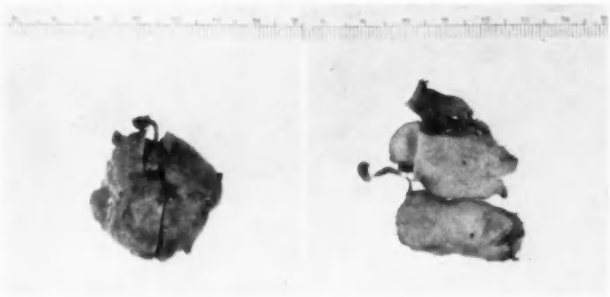


FIG. 2 — (Frost) — Tumor removed from the left orbit. (December 2, 1935)

Operation was performed on December 5, 1935. Incision through the skin and fascia of the upper lid exposed a circumscribed tumor mass of distinctly olive green color. The mass was easily separated from the surrounding tissues. It was somewhat adherent to the periosteum of the roof of the orbit and the supraorbital ridge, but not a part of it. It was not adherent to the sheath of the superior rectus muscle. The latter portion occupied the normal situation of the lacrimal gland. The tumor in the right orbit also was removed. This was less than half the size of the one on the left, and did not include any of the lacrimal gland area. Palpation of the orbit through the incision revealed no evidence of other tumor masses farther back in the orbit, and when the sutures were in place, there was little displacement of either eyeball (Fig. 2).

The tumor from the left orbit measured 3x3x1 cm., was convex against the roof of the orbit and somewhat concave in its lower part to fit the contour of the globe. The gross examination of the tumor and frozen sections established the diagnosis of chloroma (Fig. 3). It was firm and of a dirty green color. It had an elasticity suggestive of fibrous tissue proliferation. Its contour was fairly smooth, but the mass was not covered with a membrane, nor was it encapsulated. Section of the growth showed a fibrous tissue background infiltrated with round cells. There was a large portion of lacrimal gland attached to and associated with one margin of the tumor. The glands were quite normal in appearance, with only slight cloudy swelling presumably caused by some vascular disturbance. The process appeared to be one of extension into, rather than of primary involvement of the lacrimal gland.

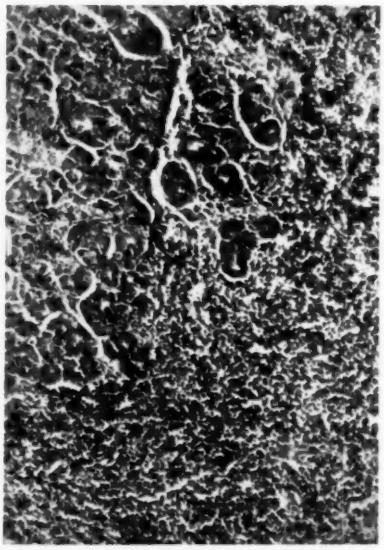


FIG. 3 — (Frost) — Section of tumor removed from the left orbit, showing invasion of the lacrimal gland. (× 150)

After the operation, the child's general condition was satisfactory except for a slight elevation of temperature daily in the afternoon to 100.5° F. There was constant drainage of blood and serum from the incisions which required changing of dressings each day, but there was no infection in the wounds. Nevertheless, the child displayed progressing weakness with a rapid drop in the number of red blood cells and in hemoglobin content. A transfusion of 250 cc.

of citrated blood six days after the operation was followed by a chill with a temperature rise to 104.5° F. Later the temperature fluctuated between 99 and 100°. The oozing from the wounds ceased and they healed promptly and there was much improvement in the patient's general condition, except for an occasional complaint of pain in the eyes. By December 18, the boy was able to be up and play about the ward, at which time the admission weight of 32 pounds was still maintained.

On December 24 he was allowed to leave the hospital for the Christmas holidays, on condition that he return. Except for weakness and anemia, his general condition was good. There was no evidence of recurrence or new tumors. Between January 14 and

Fig. 4 — (Frost) — Photographs of patient. (February 14, 1936)
Note right facial paralysis.

January 22, the patient received a course of roentgen therapy over the parietal regions. A total of 2400 roentgen units was administered in eight applications. There was little change in his appearance and condition during the month of January. His appetite was good and his weight was maintained. He did not return to the hospital until January 27, 1936.

Following a transfusion on February 5 of 375 cc. of citrated blood into the jugular vein, the child contracted chicken pox and was transferred to the Isolation Hospital where he remained until February 18. On February 14 a mass was palpated beneath the lower lid of the right eye. This grew rapidly. A smaller mass also was palpated in the same position in the left orbit (Fig. 4). Right

facial paralysis and deafness, along with some swelling in the right temporal region and cheek were discovered, but these symptoms improved after irradiation therapy.

About this time the patient began to complain of severe epigastric pain which was relieved only when he sat upright. The tumor mass in the right orbit enlarged rapidly. Because of increasing exophthalmos and severe pain, the mass was removed through an incision in the lower lid on February 20. This growth measured 3x2½x2 cm., and was pathologically identical with those that were removed earlier (Fig. 5). Bleeding and oozing through the dress-

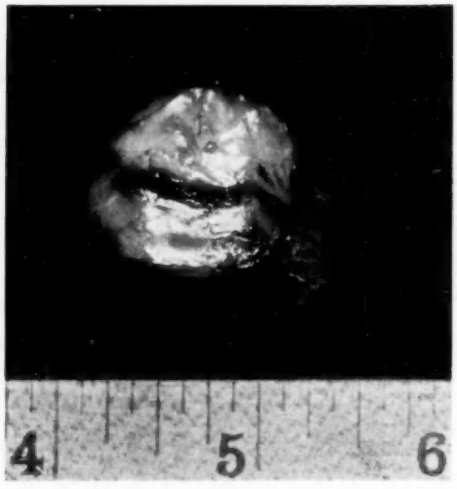


FIG. 5 — (Frost) — Tumor removed from the floor of the right orbit. (February 20, 1936)

ings was continuous. The patient's temperature rose to 105° and death was expected. The day after the operation, 150 cc. of blood were withdrawn and 350 cc. of the father's citrated blood immediately supplied. The next day the patient's condition was so much improved that the procedure was repeated three days later. A single treatment with roentgen rays (600 r) was administered on February 29.

The tissues about the wound became swollen and edematous from a large hematoma. There was no healing nor granulation and the tissues became gangrenous beneath the skin of the cheek and temple. About two weeks after operation, the patient was taken to

the operating room and the wound was thoroughly cleansed and irrigated. The patient's condition improved after this and his temperature dropped to between 99° and 100° F. There was less discharge from the wound and the patient was much more comfortable.

The tumor mass previously palpated in the floor of the left orbit had disappeared, probably as the result of irradiation, but the exophthalmos increased gradually. Numerous lesions then appeared in the form of skin nodules which later broke down into abscesses of the chin, forehead and outer canthus of the left eye. At first these appeared firm and nodular, but rapidly became necrotic, punched out ulcers discharging foul purulent material. No biopsy or cultures were made of these, but they probably were not chloroma. There was marked swelling of both anterior and posterior cervical lymph nodes; one of these was removed for biopsy.

The right temporoparietal region displayed increased thickening, apparently due to elevation of the periosteum and increased thickness of the bone, but repeated roentgenograms showed no evidence of bone change throughout the patient's entire course.

About three weeks after the last operation (March 12, 1936) the patient complained of severe pain in the abdomen and the next day there was marked abdominal distention which was not relieved by an enema or other measures. The output of urine was scanty and there were no stools. Four days later the distention was somewhat relieved by involuntary stools and urination. This was accompanied by rectal prolapse and bleeding. Coughing increased, the child's breathing became labored and his temperature rose to 104°. His condition became progressively worse during the few days until his death on March 19, 1936.

Necropsy — The child's body was examined after death and revealed the following abnormal findings: The soft tissues around the right eye were ecchymotic, swollen, ulcerated and injected. There was slight edema and ecchymosis of the cornea. No nodules were palpable on the bones. The mesenteric lymph nodes were slightly enlarged. The left thoracic cavity contained a slightly increased quantity of fluid. There were adhesions between the posterior surface of the left lung and the vertebral column. They were firm and represented the growth of a tumor from the vertebral column into the thoracic cavity with extension to the lung. The sternum showed a slight bluish nodularity on the left side, extending from the first rib into the first interspace and adjacent to the

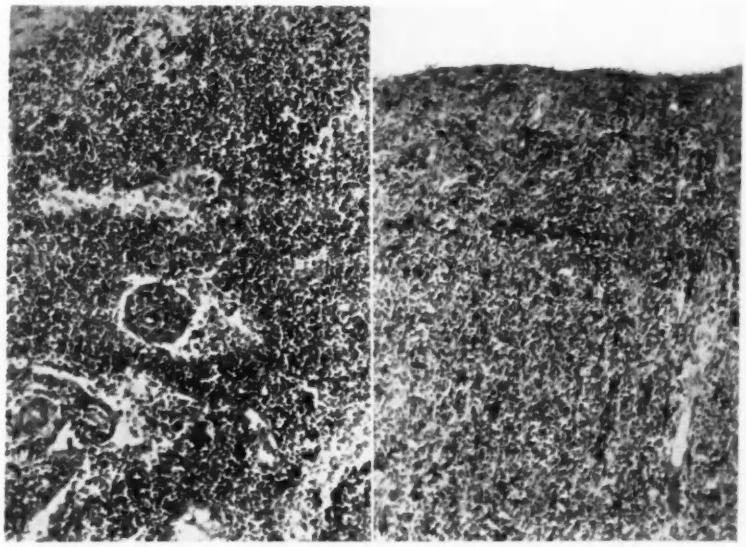
periosteum in this region. The spleen weighed 110 grams and there were some small accessory spleens at the hilus. The left kidney weighed 110 grams and the right kidney 80 grams, but neither showed anything remarkable on cut section. The peripancreatic lymph nodes were markedly enlarged.

The bone marrow in the left femur was examined; it was hyperplastic, grayish-red in color, and of a semi-necrotic type. There was a heavy infiltration of a greenish-yellow tumor over the vertebral periosteum at the approximate level of the third thoracic vertebræ, with extension downward to the mid-lumbar region. The growth extended over the left border and the side of the vertebra and into the thoracic region and stood out over the adjacent portions of the ribs. On cut section the tumor was very firm, of a dirty yellow-green color with foci appearing hemorrhagic and necrotic. The appearance of this tumor was identical with that primarily removed from the left orbit and also the right orbit. The dissected vertebræ showed apparent extension through the neural foramina into the vertebral canal with compression of the spinal cord. This pressure was exerted from a posterior position, since none of the tumor tissue was anterior to the cord. The dura of the canal appeared to be intact and showed no evidence of penetration by the malignant growth into the subdural space.

Microscopic Examination of Tissues — The characteristic feature of the case was that of subacute myelogenous leukemia, with marked primary hyperplasia of the bone marrow and infiltration or hyperplasia of primitive cells of the granulocytic series, particularly in the periosteum, to a moderate degree in the lymph nodes, and with no infiltration into organs most commonly involved in myelogenous leukemia, such as the liver, kidneys and pancreas. The peculiar dirty grayish-green character of the tumor, its predilection for periosteum and the established myelogenous leukemia placed it in the class of tumors called chloroma (Fig. 6).

The patient's symptoms were directly dependent upon the disturbance of function by growth of the tumor in organs in immediate proximity to the sites of invasion of the periosteum in the orbits and spinal column. Death was due to disturbance of metabolism, paralysis of compression myelitis, terminal septicemia and lobular pneumonia.

Hematologic Study — After the diagnosis was established by the blood count and the biopsy of the orbital tumors, the patient was placed under the care of Dr. B. K. Wiseman of the Depart-



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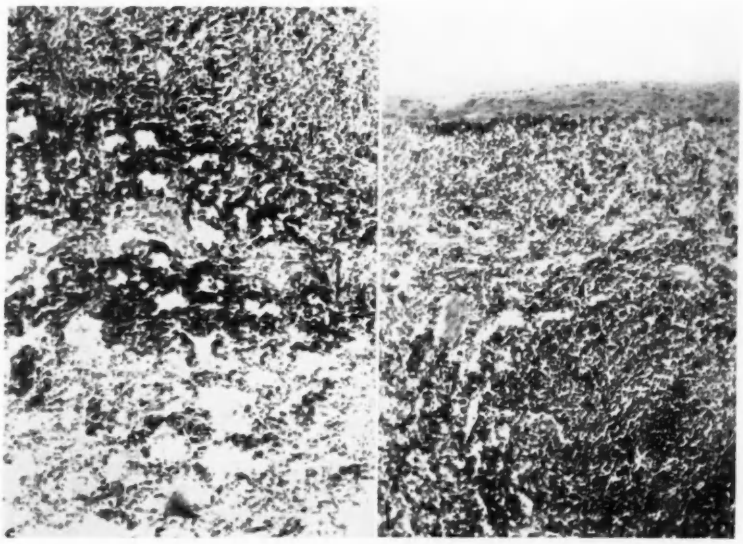

C D

FIG. 6 — (Frost) — Sections of (a) lymph node, (b) periosteum, (c) lung, and (d) spleen. (× 150)

ment of Medical Research who made a thorough study and has tabulated the blood findings in the accompanying chart (Fig. 7).

In common with most of the previously reported cases of chloroma and of acute myeloid leukemia, the blood picture of this patient shows the dominance of relatively immature cells of the myeloid series. While the white blood cells of the various types fluctuated in numbers from time to time and in response to various therapeutic procedures, for the most part myelocytes of the very young, or "A" type represented about one-half of the circulating white cells. Unlike most cases of acute myeloid leukemia, however, myeloblasts in large number appeared only infrequently and did

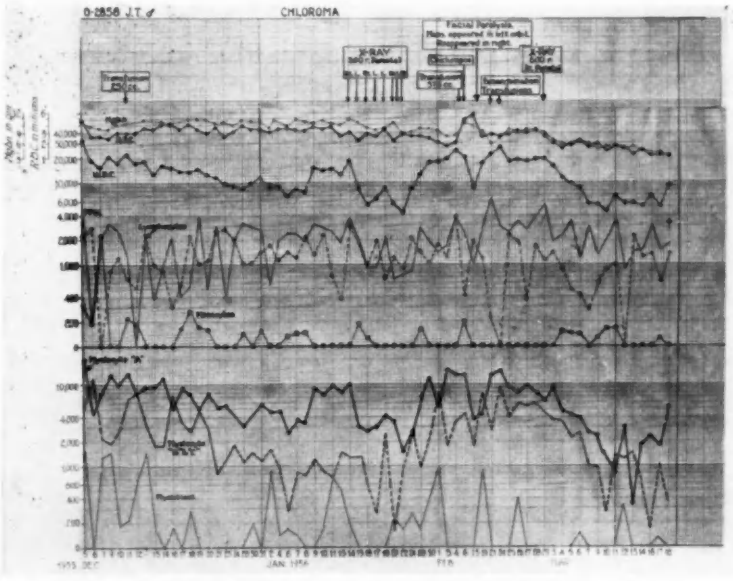

FIG. 7 — (Frost) — Chart showing complete blood picture during patient's entire course. Note particularly the fluctuations following blood transfusion and roentgen therapy.

not tend to increase either in relative or absolute numbers as the disease progressed. In common with leukemia in general, a marked anemia and thrombopenia was shown throughout the course of the disease.

The response of these circulating elements to certain therapeutic procedures and circumstances that supervened during the course of the study of this patient are shown in figure 7. On December 11, after transfusion of 250 cc. of citrated blood, there was a gradual but definite fall in the total white blood cell level

from more than 20,000 to approximately 8,000. The reduction in cells consisted chiefly of the young myeloid cells. The fall in the myelocyte A, B and C type, and particularly the myeloblasts, was outstanding. With the diminution in young myeloid cells, there was a gradual rise in mature neutrophiles and lymphocytes. This remission lasted until approximately January 6, at which time there was an increase in the total number of white blood cells and in immature myeloid cells.

On January 13, deep irradiation was administered over the right parietal region, with reduction in total white blood cells and myelocytes of much less magnitude than that following the blood transfusion. Immediately after discontinuance of roentgen therapy, the white count started to climb, with a particularly prominent increase in immature cells, myeloid series.

The patient contracted a mild case of chicken pox on February 6. Little change in the blood picture followed this infection, which is of interest since infections in the course of myeloid leukemia often are followed by temporary marked remissions in the basic leukemic state.

Following exsanguination transfusions which were given on February 21 and 24, the white blood cell count fell gradually from about 20,000 to an approximate level of 5,000, with a marked diminution in young myelocytes and myeloblasts. About March 10, there was a recrudescence in the activity of the disease, with accompanying rise, particularly in myelocytes A, until death supervened.

The type of cell involved in this case had the usual characteristic signs of immature myeloid cells. Some of the myelocytes had two nuclei and many had multiple nucleoli, often of very large size. A large proportion of these cells contained hyalin rods, known as Auer bodies, in the cytoplasm, some of which took the neutral red stain, and others rejected this dye. In the preparation stained by Wright's technic these rods stained to the same quality and color as the granules of the neutrophils. The significance of these bodies is not clear, but it is apparent that they occur only in the myeloid cells of the myelogenous type of leukemia. They are never seen in infectious states or in blood dyscrasias other than myeloid leukemia, in which there is a severe shift to the left in the myeloid series. The Auer body phenomenon persisted during the entire course of observation of this patient. The lymphocytes and monocytes did not show any qualitative changes whatsoever except for a mild shift to the left. The total number of lymphocytes and monocytes was, for the most part, always within the range of normal.

Oxydase stain upon the cells of the peripheral blood showed a positive reaction in 85 per cent, and negative in 15 per cent. A supravital differential count done at the same time upon the blood showed 84 per cent granule-containing cells and 16 per cent non-granule-containing cells. The latter group consisted of 15 per cent lymphocytes and 1 per cent monocytes. Hence the differentiation of cells in the peripheral count checked very closely with the number which theoretically are oxydase positive, and further substantiated the classification of cells.

The blood studies on this case were supplemented by study of a section of a lymph node from the right cervical region. This showed that some of the nodes in this chain had the cellular content within the capsular borders almost completely replaced by immature myeloid cells. One small node, however, showed only a minor degree of infiltration of the abnormal cells. There was no change in the reticulo-endothelial structures of these lymph nodes nor any alteration in the lymphocytes.

Repeated examination of the urine for Bence-Jones bodies (five observations) yielded negative results. Several observations on the fragility of the red blood cells showed a very slight increase in resistance to hemolysis by hypotonic solutions, a finding which has been consistent in the studies of myeloid leukemia in the hematologic laboratory of this hospital.

SUMMARY AND CONCLUSIONS

A typical case of chloroma in a boy, aged four years, is reported. These cases have a place in ophthalmic literature only because exophthalmos is so often the first symptom which brings the patient to a physician. Hence ophthalmologists must be familiar with the condition but should seek the collaboration of the hematologist, in whose domain this disease more properly lies.

It is evident from the lengthy discussions in ophthalmic literature of the nature of chloroma that some authors have been led far afield in the emphasis on so-called characteristic symptoms such as separation of the suture lines, which represent merely secondary manifestations dependent on the situation of the tumor growths. At the same time there has been exhibited a failure to emphasize and even lack of understanding of the underlying pathology of the disease, which is a particular type of myelogenous leukemia.

The diagnosis is made on the blood picture and the presence of tumor masses of a greenish color which contain embryonic myeloid cells.

Roentgen irradiation is the most satisfactory treatment, for it causes a reduction in the size of the tumors, and relief of pain. If the total white cell count is high, the spleen and lymph nodes as well as areas with pronounced symptoms should be irradiated, as is done in cases of leukemia. Replacement transfusions also are of value in relieving the symptoms and reducing the number of abnormal cells in the blood. Surgery should not be attempted (except for biopsy) unless the roentgen therapy has failed to decrease the exophthalmos, or to preserve the eye from destruction.

In the presence of exophthalmos with or without palpable orbital tumors, especially in children, a complete blood study with supravital staining methods should be made by a competent hematologist, in order to establish the diagnosis. A routine blood count may fail to reveal the true nature of the condition.

REFERENCES

1. Gump, M. E., Hester, E. G., and Lohr, O. W.: *Monocytic chloroma.* Arch. Ophth. 16:931-949, (Dec.) 1936.
2. Burns, Alan: *Observations on the Surgical Anatomy of the Head and Neck.* First Am. Ed. by G. S. Pattison. Lucas, Baltimore, 1823, p. 386.
3. King, A.: *Case of chloroma.* Month. J. Med. Sci. 17:97-104, 1853.
4. von Recklinghausen: *Ueber Chlorome.* Tagebl. d. Versamml. deutsch. Naturf. u. Aerzte. 58:421, 1885.
5. Dock, George: *Chloroma and its relation to leukemia.* Am. J. Med. Sci. 106:152-185, 1893.
6. Dock, George, and Warthin, A. S.: *A new case of chloroma with leukemia, with a study of cases reported since 1893.* Med. News. 85:971, 1025, 1078, 1118; 1904.
7. Lehndorff, H.: *Zur Kenntnis des Chloroms (Chlorleukæie, Chloroleukosarkomatose).* Jahrb. f. Kinderh. 72:53-77, 1910.
8. Gould, E. W., and LeWald, L. T.: *Chloroma: a report of two cases, together with the roentgenologic findings.* Am. J. Obst. 73:1159-1162, 1916. Also, Arch. Ped. 33:417-433, 1916.
9. Brannan, Dorsey: *Chloroma: The recent literature and a case report.* Bull. Johns Hopkins Hosp. 38:189-216, 1916.
10. Feer, E.: *Zur Kenntnis der Chloroleukämien im Kindesalter.* Jahrb. f. Kinderh. 116:155-164, 1927.
11. Washburn, A. H.: *Chloroma: Report of a case with recovery following roentgentherapy, with a review of the literature.* Am. J. Dis. Child. 39:330-348, 1930.
    ——Personal Communication.
12. King, Clarence: *Case of chloroma with orbital involvement locally benefited by x-ray therapy.* Tr. Am. Ophth. Soc. 32:340-353, 1934.
13. Merculov, I. I.: *Chloromatous neoformations and their roentgenotherapy.* Russkii Ophth. J. 7:621-630, 1928.
14. Gamble, R. C.: *Chloroma.* Illinois Med. J. 52:466-471, 1927.
15. Boots, R. H.: *Chloroma with report of a case.* J. Lab. Clin. Med. 2: 622-631, 1916-17.

DISCUSSION

Dr. T. L. Terry, Boston: Exophthalmos is almost invariably present in chloroma and it is often the first symptom, as the essayist has stated. Granting that leukemia is relatively rare, it is very important that leukemia with or without the green tumor should be considered in the differential diagnosis of unilateral or bilateral exophthalmos. There is very little reference to chloroma or leukemia as a cause of exophthalmos. In Spaeth's recent article (Arch. of Ophth. 1937) chloroma is mentioned, but in the symposium before the Academy in 1934 Plummer and Wilder state that exophthalmos in myelogenous leukemia is almost unknown.

That leukemia must be considered in the differential diagnosis of the causes of exophthalmos is stressed in my mind by the following case. The patient was referred to me by an internist. The internist had already made a careful examination including blood study, X rays and basal metabolism. The blood study showed a mild anemia, 8,000 white blood cells, and a normal differential count. The left eye was 7 mm. more prominent than the right eye. There were some small bright red rounded hemorrhages in the retina near the disc in each eye. Because of the bilateral retinal hemorrhages it seemed that the cause of the trouble must be a general disease. I repeated the blood count and found many immature granulocytes and 16,000 white blood cells per cubic millimeter of blood. From subsequent studies the white blood count was found to have increased rapidly to 49,000 within a week. It was evident that the blood picture was rapidly changing from an aleukemic phase to marked myelogenous leukemia. The exophthalmos disappeared almost completely in two days following X ray therapy to the orbit (⅔ erythema dose). In spite of a great reduction in the white count by general X ray treatment the patient became weaker and died within the month. Since no biopsy was done and no post mortem examination was made, the color of the orbital tumor was not determined and for that reason a positive diagnosis of chloroma is impossible.

Had it been suspected that the exophthalmos might have been due to a leukematous disease during the aleukemic phase, very diligent and repeated blood examinations with supravital stain might have given the diagnosis much earlier. According to Isaacs and Danielan (Amer. Jour. Med. Science, 174:70, 1927), the abnormal white cells are largely thrown out of the body into the intestinal tract rather than meet destruction in the body, and it follows that the ratio of normal to abnormal white cells in the blood and in the saliva is inversely related. Use may be made of the saliva cell count in relation to the blood count in detecting leukemia during the aleukemic phase. Allen and Dickey (Amer. Jour. of Roentgenology, 38:57, 1937) consider the correlated study of saliva cells and blood cells of value in regulating the dosage of X ray therapy.

The great difference between chloroma and myelogenous leukemia is the development of a green tumor in the former. The significance of the green tumor may not be known until the cause of the green color is fully under-

stood. There have been many attempts to explain the color, as the essayist has mentioned. Thomas and Bigwood (Compt. rend. Soc. de Biol., 118:381, 1935) feel that the green color is due to porphyrin, since they have shown by spectroscopic study the presence of free porphyrin in the tumor. They have recovered porphyrin from the tumor by extraction with glacial acetic acid and ether.

Why do these green tumors have a predilection for growth in orbital tissue? The orbit is remarkably free from lymph nodules. The orbit must certainly have lymphatic channels which have little or no connection with the lymphatic channels of the face and neck. It is possible then that the very lack of free communication of lymph channels to the face and neck may tend to produce a physiological lymph stasis in the orbit. Such a condition would favor development of myelogenous, chloromatous, or lymphomatous nodules.

The appearance of white centers in retinal hemorrhages is presumably diagnostic of leukemia although it is not invariably present. It is due to accumulation of abnormal white blood cells in the center of the hemorrhage. I should like to ask Dr. Frost if this is also observed at times in chloroma?

Dr. Georgiana Dvorak-Theobald, Chicago: Dr. Frost's study of this case has been most complete, and his blood findings and histologic pictures of great interest.

Chloroma is a rapidly growing malignant neoplasm, involving the hematopoietic system, with metastases of cells to distant organs and associated with a leukemic blood picture. It is almost invariably fatal, with a duration of 3 to 5 months following recognition.

Up to the present time, about 200 cases have been reported in the literature.

The most characteristic sign is the predilection of the tumors for the periosteum of the cranial bones, especially of the orbit.

The rapidity of growth and extent of the orbital tumors may give rise to a number of ocular symptoms, namely: exophthalmus, drying of the cornea (due to extreme proptosis), optic neuritis, retinitis, sluggish reactions of the pupils, anisocoria, mydriasis, conjunctivitis, cataract, exotropia and nystagmus. In one type of chloroma the aural structures are affected.

Etiology: The tumors apparently arise from localized growth of leukemic deposits or metastases, and the tumor cells metastasize in the blood to produce leukemia.

In about half of the cases the periosteum of the orbital bones is the site of predilection. Why this is so is not known.

Differentiation between chloroma and myeloma is based on the usual blood picture of leukemia and the green colored tumor in chloroma, and the tendency to spontaneous fracture in myeloma. As a rule the Bence-Jones type of protein is present in the urine of myeloma patients and only rarely in chloroma patients.

It is thought that the green color of chloroma is due to a lipochrome, or that it is due to a blood pigment; but nothing is definitely known. Roehn and his associates say that it is due to phagocytosis of the blood in obstructed capillaries by endothelial cells.

Histologic pathology: The tumor cells are usually large undifferentiated mononuclears, designated as myeloblasts or lymphocytes. Indeed, in the past, pathologic differences of opinion have centered around the lymphatic or myelogenic origin of the neoplasms. In a case reported by Roehn, Riker, and Olsen (Ann. Intern. Med. 10:1054, (Jan.) 1937), pathologic examination of the tumor by Dr. C. V. Weller of the University of Michigan showed that it was neither myelogenic nor lymphatic in origin, as the tumor cell is a *primitive blastoma which might conceivably differentiate into either a lymphocyte or myelocyte.*

The possibility of tumor cells having a double potentiality of development is well brought out in Berger's theory of the "double potentiality of development" of a single neuropigmental blastoma. That is to say, that the cells of the blastoma are capable of evolving either as elementary ganglion cells or Schwannian melanic elements, "for the Schwann nerve cells and the melanic cells there is a *common primary blastoma* the elements of which become neoplastic."

Gump et al. (Gump, Hester, and Lohr: Arch. Oph. 16:931, (Dec.) 1936) treat of monocytic chloroma, which they say is usually classed as a distinct entity; but the origin of the monocyte whether from reticulo-endothelial elements or not is doubted. In their case, autopsy sections of the tissue showed cells of the neoplasm in a connective tissue reticulum, satisfying the criterion for true blastomatous neoplasm of the endothelial system.

In an early article, more than 20 years ago, Dock and Warthin (Med. News (New York), 85:911, 1904), who made an exhaustive study of chloroma, concluded that "chloroma is a tumor-like hyperplasia of the parent cells of the leukocytes primarily in the red marrow, the periosteum being involved."

I have seen two cases of chloroma; one with Dr. Cupler at St. Anthony's Hospital. This patient had a blood picture similar to that described by Dr. Frost. The orbital contents were sent to me after death of the patient, but are not ready for study. The second case is being successfully treated by irradiation over a period of several years by Dr. Poucher. The eyes are proptosed, but the lids have grown sufficiently to properly protect them. The proptosis is now due to a shallow orbit. The vision is normal, and the fundus shows no changes.

Dr. Albert D. Frost, closing: I thank the discussants for their interesting remarks.

Relative to the saliva count, questioning hematologists, I believe they feel this is a very noteworthy observation but as yet a scientific curiosity, nevertheless it may be a valuable diagnostic point and should be done.

I think it unwise to indulge in a lengthy discussion of the difference in color, since this is a much disputed point even among hematologists, who find, for instance, a decomposed product of red cells which seemingly are rapidly destroyed in this disease. The definite anemia present, therefore, seems to be a satisfactory explanation of the phenomena. However, it is also plausible that the reaction may be due to the granule content of the predominating myelocytes, such as was mentioned by Boots.

I have not observed retinal hemorrhages in this case, nor do I find any discussion of such in the literature. Most myeloid leukemias, as I understand it, do not show the characteristic retinal hemorrhages, and they are prevalent in the lymphatic leukemias. A probable explanation may lie in the fact that in lymphatic leukemia there is more definite thrombopenia than there is in the myeloid type.

I might add that the autopsy did include a rather thorough examination of this boy's one eye, and we have never had a better section of a normal eye throughout.

EPITHELIUM IN THE ANTERIOR CHAMBER OF THE EYE AFTER OPERATION AND INJURY

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NEW YORK

IMPLANTATION or down-growth of epithelium into the anterior chamber of the human eye following perforating injuries or intraocular operations is a well-known clinical entity. This serious complication often results in loss of the eye from an intractable secondary glaucoma.

A survey of the literature reveals that this epithelial invasion of the interior of the eyeball produces three different types of lesions —

1. "Pearl" tumors of the iris;
2. Post-traumatic cysts of the iris;
3. Epithelization of the anterior chamber.

Iris cysts and "pearl" tumors have been described following perforating injuries and intraocular operations. These new-growths may develop after the implantation of a cilium into the anterior chamber, presumably as a result of the proliferation of the epithelium of the follicle, adjacent glands, or skin. Such epithelial structures may be formed in the eye within a period of from several months to many years. Collins[1] in 1891 described a large epithelial implantation cyst in a shrunken globe twenty-eight years after the injury. Many authors have presented cases of post-traumatic or postoperative cysts of the iris. Among them may be mentioned Mackenzie,[2] von Graefe,[3] de Wecker,[4] Hulke,[5] Knapp,[6] Rothmund,[7] Monoyer,[8] Feuer,[9] Sattler,[10] Stolting,[11] and others.[12]

The general subject of iris cysts has been thoroughly discussed by Rothmund,[13] de Wecker,[14] Hosch,[15] Greef,[16] Collins,[17] Urmetzer,[18] and, more recently, Ginsberg,[19] and Villard and Dejean.[20]

Epithelization of the anterior segment of the eyeball as a consequence of corneal injury or intraocular operation has been pre-

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sented in a number of case reports by Guaita,[21] Meller,[22] Elschnig,[23] Ginsberg and Hirschberg,[24] Collins,[25] Hagen,[26] Berg,[27] Strader,[28] Balacco,[29] Salus,[30] Levine,[31] Marcove, [32] and many other authors. An interesting form of this condition occurs as an hourglass cyst of the iris or anterior chamber, as exemplified in the patient reported by Critchett.[33] The slit-lamp and corneal microscope are valuable in the diagnosis of these cases.

The development of an epithelial cyst of the anterior chamber is a serious complication of any intraocular operation, especially of those procedures in which a wide opening is made into the interior of the anterior portion of the globe, i.e., operation for cataract. Once the epithelization of the anterior chamber has taken place, the eye is almost always lost as the result of a stubborn glaucoma. Recent attempts to destroy or inhibit the growth of the epithelium in the anterior chamber by the use of roentgen-rays or radium have been encouraging (Axenfeld,[34] Jendralski,[35] Handmann,[36] Custodis,[37] Vail,[38] and Federici[48]).

The following case of postoperative cyst of the anterior chamber was successfully treated by the use of roentgen-radiation:

CASE REPORT

Gertrude B., a 61-year-old American practical nurse, was first seen in January, 1934, on the service of Dr. D. B. Kirby at the Institute of Ophthalmology of the Presbyterian Hospital, with a history of life-long high myopia, and recent failure of vision. Examination showed bilateral nuclear and cortical lenticular opacities, contracted visual fields and normal intraocular pressure. The vision of the right eye was 20/100 with lens S. —20.00 ⊂ cyl. —1.50 ax. 165. The vision of the left eye was perception of hand movements at 6 inches, and could not be improved by glasses. The interior of each eye revealed fluid vitreous, many vitreous opacities, and extensive areas of chorio-retinitis. A preliminary iridectomy was performed on the right eye in November, 1934, with uneventful healing. Two months later, an intracapsular cataract extraction was done; this was followed by incarceration of the temporal iris pillar in the operative wound, a mild uveitis, and prolapse of the vitreous into the anterior chamber. By March, 1935, vision of this eye was found to be 20/30 with —4.50 cyl. ax. 130. In May, 1935, the patient returned, complaining of photophobia. Examination of the congested right eye at this time showed a cyst lying between the temporal pillar of the iris and the posterior surface of the cornea, and extending through the corneosclera at the limbus (Fig. 1).

FIG. 1 — (Perera) — Photograph of right eye showing postopera- cyst in the anterior chamber (marked with arrow), and congestion.

The cyst measured 2½ by 3 mm. The intraocular tension was 23 mm. Hg. (Schiötz tonometer). While under observation, the intraocular cyst slowly increased in size and radiotherapy was advised. The patient was referred to Drs. H. H. Kasabach and O. L. Henderson, of the Radiotherapy Department of Presbyterian Hospital for treatment. The right eye was treated with five doses of 150 r. at weekly intervals until a total of 750 r. had been given, using 200 K.V.P., 0.5 mm. copper filtration, and 50 cm. T.S.D. When examined in June, 1935, the cyst had shrunk to a light yellowish mass. In August, 1935, a slight reformation of the cyst had taken place,

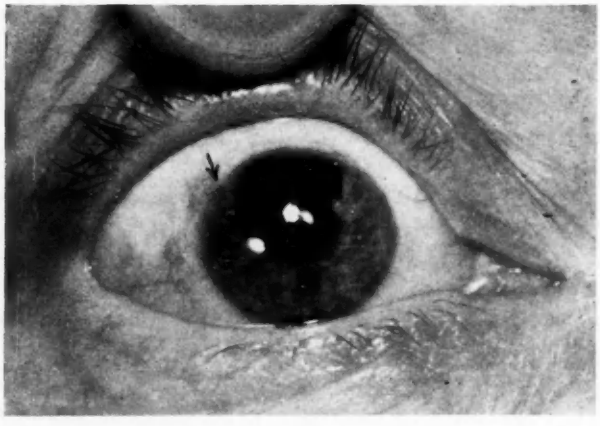

FIG. 2 — (Perera) — Photograph of right eye showing shrinkage and disappearance of cyst of anterior chamber following roentgenradiation (previous site of cyst marked with arrow).

and a second series of five treatments was given, using 108 K.V.P. and 3 mm. aluminum, with 25 cm. T.S.D. The patient's right eye received a total of 1550 r. in the two series of treatment. By the middle of October, 1935, the cyst had disappeared (Fig. 2). When the patient was examined in August, 1937, there was still no evidence of a cyst in the anterior chamber of the right eye.

The files of the Pathology Department of the Institute of Ophthalmology of the Presbyterian Hospital during the period between 1929 and 1937 contain records of eight eyeballs removed because of intractable glaucoma due to epithelization of the anterior chamber. Of these globes, the "anterior chamber cyst" followed perforating injuries in four cases, and cataract extraction in the remaining four instances. It is interesting to note that the latter group represents over one-ninth of the thirty-five globes in the Institute collection which had been removed during this period following a previous cataract operation. One of these eyes contained an hourglass epithelial cyst, the intraocular cyst communicating with a large subconjunctival cyst; the destructive glaucoma which compelled enucleation in this case had been preceded by a long period of postoperative hypotony. Careful microscopic studies of the four postoperatively removed eyes show incarcerated iris or lens capsule or both in each case.

The factors involved in the proliferation of epithelium in the anterior chamber following injury or operation are but little understood. Although the anterior chamber is opened frequently, a localized or generalized epithelization of this space occurs only rarely. In the surgical treatment of twenty eyes suffering from absolute glaucoma, Gradle[39] made use of a conjunctival drain of the anterior chamber, and had not found any evidence of proliferation of the epithelium. Paracentesis of the anterior chamber is a common procedure, and the opening into the eyeball thus made is often reopened on subsequent days, and yet epithelium does not invade the anterior chamber in these cases. One eye in the Institute collection contains degenerating corneal epithelium on the posterior surface of the cornea adjacent to a recent paracentesis wound. It is difficult to explain why epithelium proliferates in the anterior chamber in some instances and not in others.

Associated with this problem is the simpler one concerning the mode of entrance of the epithelial cells. In many cases, and certainly in those following the implantation of a cilium into the anterior chamber, epithelial cells have been bodily transported from outside the eye into its interior. The Institute collection contains a

globe, enucleated soon after a perforating injury, with a mass of implanted epithelium lying upon the surface of the iris. In other cases, epithelium has invaded the anterior chamber by direct growth and extension from the outside through an opening into the eye. This is very evident in the cases with hour-glass cysts, and has been demonstrated in serial sections of a number of eyes which have been removed because of absolute glaucoma from epithelization of the anterior chamber.

A small group of workers has investigated the problem from an experimental viewpoint. Fuchs[40] discussed the tendency of corneal epithelium to penetrate a corneal wound in his Bowman Lecture in 1902. Weinstein,[41] in an experimental study of the healing of perforated incised wounds of the cornea in rabbits, described his findings at varying intervals following section of the cornea with a Graefe knife. He demonstrated the down-growing plaque of epithelium which was gradually pushed forward by new-formed connective tissue, and presented the evidences of regeneration of Descemet's membrane.

The first experimental work on implantation of epithelium into the anterior chamber was reported by Dooremaal[42] in 1873. This author, using dogs and rabbits, placed inert and non-living foreign bodies, hair, conjunctiva, oral mucous membrane, and skin upon the iris through a narrow corneal wound. The mucous membrane from the lip formed a tumor similar to a pearl cyst, or epidermoid tumor.

In the following year, Goldzieher[43] performed a series of experiments on rabbits, implanting various substances into the anterior chamber through a small incision at the corneal limbus, made with a lance-knife. He placed conjunctiva, nasal mucosa, a segment of a peripheral nerve, a portion of another rabbit's cornea, and a Fallopian tube into the anterior chamber. A cyst formed in the case of the implantation of nasal mucosa. In the experimentally implanted corneal segment, the author found that the alien tissue diminished in size, and that, when the eye was removed on the thirtieth day, the surface of the foreign tissue was still covered with epithelium. Goldzieher divided the fate of implanted tissues into three categories: encapsulation; growth — then shrinkage; and proliferation.

The next investigator to attack this problem was Schweninger,[44] who presented his experiments on the transplantation and implantation of hairs in 1875. He performed two series of investigations, — transplanting hairs upon granulation tissue, and in-

troducing hairs through a corneal puncture into the anterior chamber of rabbits and dogs, placing the root of the hair upon the iris. He found that hairs produced no reaction in the anterior chamber. In most cases the hair-root united firmly with the iris, often with proliferation of the hair-follicle cells. Occasionally the hair fell out of the iris, leaving follicle cells behind.

Hosch[45] performed some experimental studies on iris cysts in 1885, making use of rabbits. He made a small limbal incision with a keratome, and placed the foreign body upon the iris with forceps, a fine silver sound, or a needle. He implanted cilia and hairs in thirteen animals. Among this group, epithelial cells were never found in the iris, although there was an occasional mass of connective tissue and degenerative material. In six cases in which conjunctiva had been implanted, two showed no evidence of a foreign body after forty-three and forty-seven days, respectively. The other four were found to have a tumor formation on the iris, with new-formed blood vessels. Skin was implanted in four eyes. Of these, one became infected. In the remaining three, epithelial-lined cysts, with atheromatous contents, were formed. The author believed that the formation of iris cysts around implanted cilia was due to proliferation of the cells in an accompanying minute particle of skin. In this work he quoted Monoyer, Rothmund, Roth, de Wecker, Dooremaal, Goldzieher, Schweninger, and Masse.

Experimental work on the proliferation of epithelium in the anterior chamber was reported by Corrado[46] in 1931. In this paper he presented a case, and described his experiments on rabbits in five series. In the first, he introduced a conjunctival flap, still attached to the cornea, into the anterior chamber through a marginal incision. In these eyes, there was a tendency for the flap to be expelled from the anterior chamber. In those cases in which the conjunctival flap remained within the eye, there was complete absorption in the course of time. In the second series, the author introduced a corneal flap containing stroma and epithelium into the anterior chamber through a small incision. In this group, there was no evidence of proliferation, and the flap gradually disappeared. Even after traumatizing the eye, and attempting to maintain hypotony, the results were identical. In the third series of experiments, Corrado introduced a corneal flap into the anterior chamber, and placed a celluloid plaque between the lip of the corneal wound and the flap, removing the celluloid after fifteen days.

In these cases, the flap of tissue was found folded in the anterior chamber, completely preserved, and adherent to the iris. In the fourth group of animals, a corneal flap was placed upon the anterior capsule of the lens. Proliferation took place in almost all. In the last experiments, Corrado injured the lens to see if the lens epithelium would grow, but this did not occur. His conclusions were: It is not enough for a flap of epithelial tissue to be placed in the anterior chamber to live or proliferate. Certain conditions must be present for this to take place. Only if the corneal wound stays open for a certain length of time is it possible for corneal epithelium to proliferate in the anterior chamber. This is aided by incarceration of lens capsule or vitreous in the corneal wound. The epithelium has a tendency to grow more rapidly upon a smooth surface. Corneal epithelium in the anterior chamber is absorbed more slowly in the more hypotonous eyes. Hypotony appears to favor the vitality of the epithelium itself, possibly because the presence of albumen in the aqueous humor of such an eye makes a good culture medium.

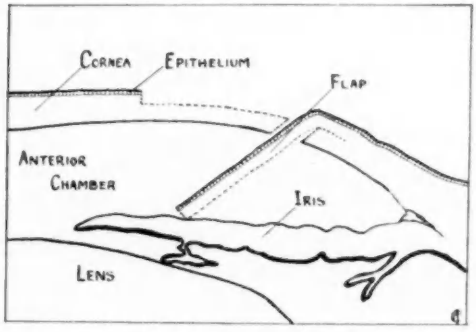

Fig. 3 — (Perera) — Sketch to illustrate operative procedure.

A recent paper by Accardi and Rossi[47] showed that replacement by new-formed connective tissue took place in the anterior chamber of rabbits following the introduction of various tissues (cornea, iris, sclera, lacrimal gland, pancreas, thyroid, thymus, suprarenal gland, liver, kidney, lung, striated muscle, dura, fascia, fat, bone, ovary, and testicle). This was true whether the transplant was homo- or autoplastic, and whether the tissue was adult or embryonic.

In an effort to obtain more data about the growth of corneal epithelium in the anterior chamber, a series of operations was performed on the right eyes of eight normally pigmented, unselected rabbits, of ages between four and six months. The left eyes of these animals were studied as normal controls for comparison.

A superficial corneal flap, including epithelium and a thin layer of stroma, was dissected from the cornea toward the limbus and pushed through a keratome incision, made beneath the flap, into the anterior chamber (Fig. 3). No intraocular structures were traumatized, nor was the iris touched by an instrument. The eyes were removed at intervals of from five days to four months, and studied microscopically by serial sections.

TECHNIC OF OPERATIVE PROCEDURE

An injection of 1.0-1.2 cc. of Pernoston was made intraperitoneally. After the animal was stuporous, it was fastened upon the operating table on its left side. The long hairs and whiskers which interfered with the operation were cut. The right eyelids and adjacent skin were swabbed with 3½% tincture of iodine. The head of the rabbit was covered with a strip of gauze soaked in 70% alcohol, exposing the operative field through a hole. The entire animal was covered with a sterile towel having an opening to permit exposure of the region of the right eye. An instillation of one-half per cent solution of Pontocain was made into the right eye, and repeated.

A speculum was inserted, and the right conjunctival sac thoroughly irrigated with normal saline solution. Fixation forceps were applied below, and a superficial corneal flap was dissected with a Graefe knife, beginning in the pupillary area and leaving the flap attached near the limbus. The corneal flap was about 4 mm. wide, and was cut by sawing movements of the knife. The superficial flap was turned back over the conjunctiva, and a keratome incision made through the cornea beneath the flap. This incision extended into the anterior chamber, and was followed by escape of aqueous. The turned-back corneal flap was gently pushed through the incision and into the anterior chamber with an iris spatula. The speculum was removed, and the lid margins sewed together with two interrupted silk sutures. A collodion and gauze dressing was applied.

CLINICAL OBSERVATIONS OF ANIMALS

In all cases, healing took place without infection. The lid sutures were removed forty-eight hours after operation.

Rabbit No. 1 was killed at the end of five days after operation; rabbit No. 2 at twenty-one days; rabbits Nos. 3 and 4 at two months; rabbits Nos. 5 and 6 at three months; and rabbits Nos. 7 and 8 at four months.

A bulging, ectatic, pigmented scar resulted in the case of rabbit No. 3. In the other animals, the corneal cicatrix was connected with the central third of the iris by a bridge of grayish tissue.

Measurements of the intraocular pressure before and after operation revealed no essential difference between the operated and non-operated eyes, the tension being between 23 and 30 mm. with the Schiötz tonometer.

MICROSCOPIC STUDY AND COMMENT

The accompanying photomicrographs (Figs. 4-15) include the pertinent findings in careful studies of the serially cut microscopic sections of the eight eyes which were operated upon, and of the eight normal eyes used as controls.

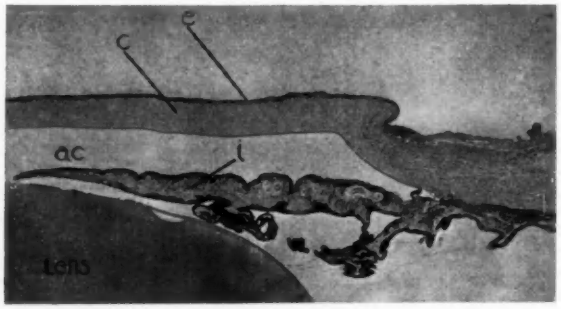

FIG. 4 — (Perera) — Section through portion of anterior segment of normal rabbit's eye, representing the left eyes of the animals used in the experiment. *c*, cornea (flattened in preparing the section); *e*, corneal epithelium; *ac*, anterior chamber; *i*, iris. (× 30)

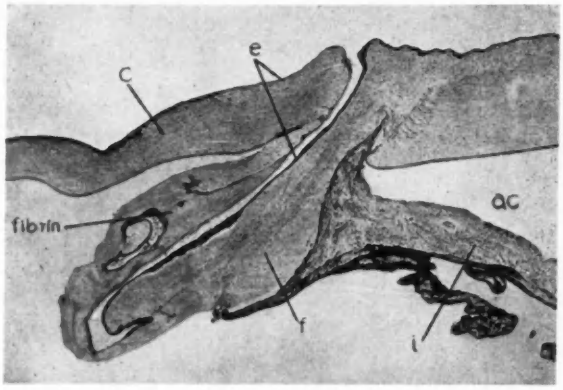

FIG. 5 — (Perera) — Section of anterior segment of the operated right eye of rabbit No. 1 (killed five days after operation). Comparing this section with the sketch of the operative procedure (Fig. 3), it can be seen that the corneal epithelium (*e*) has grown over the denuded anterior portion of the cornea in a thin layer, and has extended down upon the anterior lip of the keratome incision into and around a plug of fibrin which lies between the implanted corneal flap (*f*) and the cornea (*c*). The newly grown epithelium has formed some spaces filled with epithelial débris. A wedge of iris (*i*) tissue has become caught between the posterior lip of the keratome incision and the implanted corneal flap (*f*). The anterior chamber (*ac*) has been partially restored, although several of the serial sections showed that an epithelized fistulous tract was present. (×30)

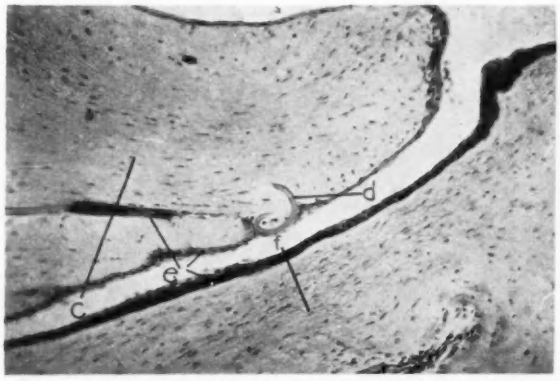

FIG. 6 — (Perera) — High power study of a detail of figure 5, showing the anterior lip of the keratome incision, and the hinged end of the implanted corneal flap (*f*). The thin layer of epithelium on the previously denuded surface of the cornea (*c*) is seen in the upper portion. This layer extends around the anterior lip of the corneal incision, forming a fistula-like opening through the cornea. A small curled loop of Descemet's membrane (*d*) is clearly outlined. The new-grown epithelium (*e*) lying upon the implanted corneal flap (*f*) is irregular in thickness. (×200)

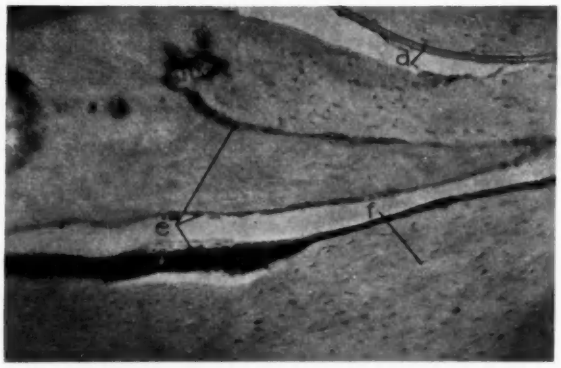

FIG. 7 — (Perera) — High power study of a detail of figure 5, showing the central portion of the implanted corneal flap (*f*) which is separated from the overlying fibrin by an epithelial-lined tract. In the upper right corner of the section can be seen a small portion of the posterior layer of the cornea with Descemet's membrane (*d*). Corneal epithelium (*e*) has invaded the fibrin plug forming small cystic spaces. (×200)

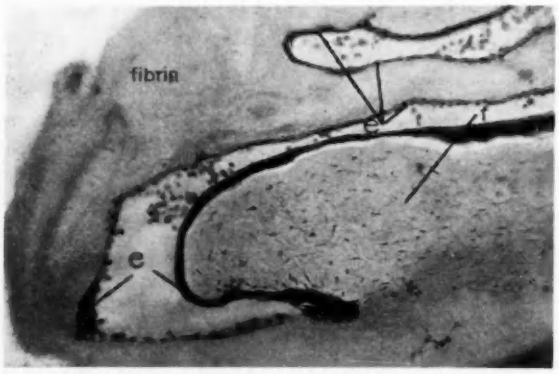

FIG. 8 — (Perera) — High power study of a detail of figure 5, showing the extremity of the implanted corneal flap (*f*) and the adjacent fibrin mass. The corneal epithelium (*e*) has extended upon the cut end of the introduced flap and has grown into the fibrin plug, forming epithelial-lined spaces, in which can be discerned cast-off epithelial cells. (×200)

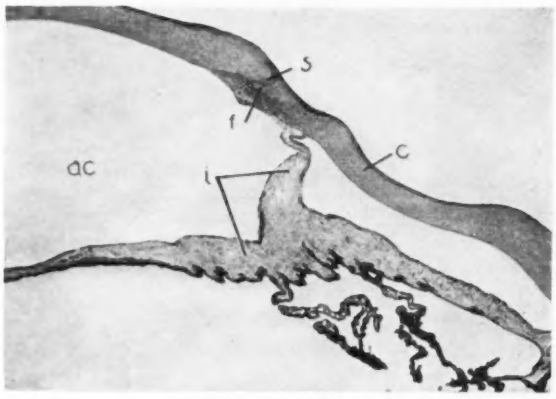

FIG. 9 — (Perera) — Section of anterior segment of the operated right eye of rabbit No. 2 (killed twenty-one days after operation). The site of the corneal incision (*s*) can be easily identified, but the implanted corneal flap has disappeared except for a small remnant (*f*). The iris (*i*) is adherent to the cornea in the region of the operation, forming an elongated anterior synechia. The anterior chamber (*ac*) is fully restored. (×30)

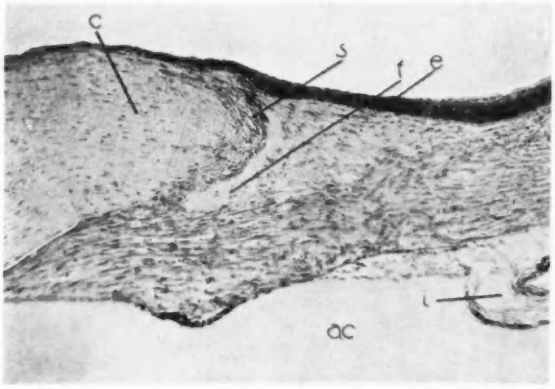

FIG. 10 — (Perera) — High power study of a detail of figure 9, through the corneal scar (*s*). The epithelium covering the previously denuded cornea (*c*) is thin. The cells forming the scar (*s*) stain more deeply than the adjacent corneal stroma, and resemble fibroblasts with a few small groups of degenerated epithelial cells. The posterior cornea shows the remains of the implanted corneal flap (*f*), and a loosely arranged fibrous tissue, the posterior portions of which contain pigment. The anterior synechia (*i*) is seen at the lower right. No epithelial cells were seen in the interior of the eye in the serial sections from which this photomicrograph was made. (×200)

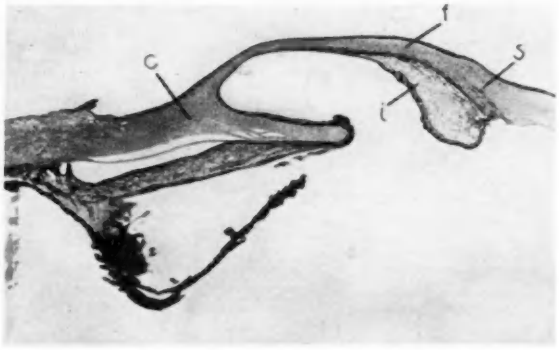

FIG. 11 — (Perera) — Section through the ectatic scar of rabbit No. 3 (killed two months after operation). The anterior lip of the keratome incision (*s*) contains fibrous tissue and pigment derived from the iris (*i*). The implanted corneal flap (*f*) has been expelled from the interior of the eye, forming the bulging and thinned area of the cornea lined posteriorly by the thinned-out remains of the adherent iris (*i*). The intraocular pressure in this eye was normal. (×30)

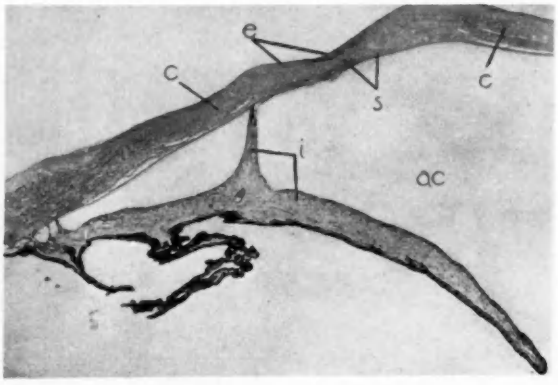

FIG. 12 — (Perera) — Section of anterior segment of the operated right eye of rabbit No. 4 (killed two months after operation). The region of the scar (*s*) is easily visible; the cornea is diminished in thickness here. Descemet's membrane is seen as a wrinkled line in the posterior part of the cornea (*c*) in the upper right corner. A fibrous layer covers the posterior cornea in this region. An anterior adhesion of the iris (*i*) extends forward posterior to the immediate site of the operative incision (*s*). (×30)

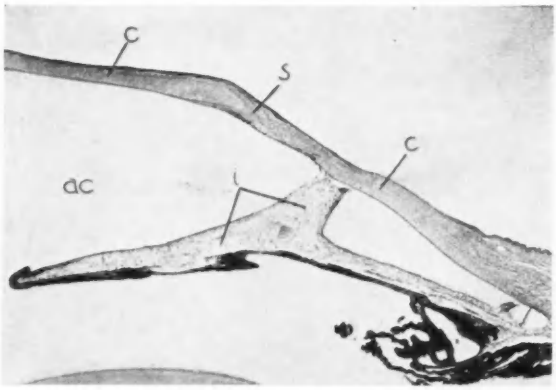

FIG. 13 — (Perera) — Section of the anterior segment of the operated right eye of rabbit No. 5 (killed three months after operation). The region of the keratome incision is seen as a scar (*s*) extending through the cornea. Pigmented fibrous remains of adherent iris lie under the endothelium on the posterior surface of the cornea beneath the scar. An anterior adhesion of the iris (*i*) is present posterior to the area of the scar. (×30)

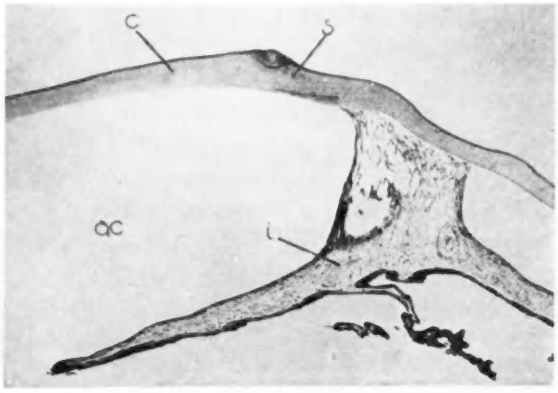

FIG. (14) — (Perera) — Section through anterior segment of the operated right eye of rabbit No. 6 (killed three months after operation). The corneal epithelium overlying the operative scar (*s*) is heaped up and irregular. In the scar there is a cystic space filled with cellular débris and lined by flattened epithelial cells. A broad anterior synechia is present, with degenerated pigmented remains of iris tissue in the posterior portion of the corneal scar (*s*). There is no evidence of corneal epithelium in the iris or in the anterior chamber. (×20)

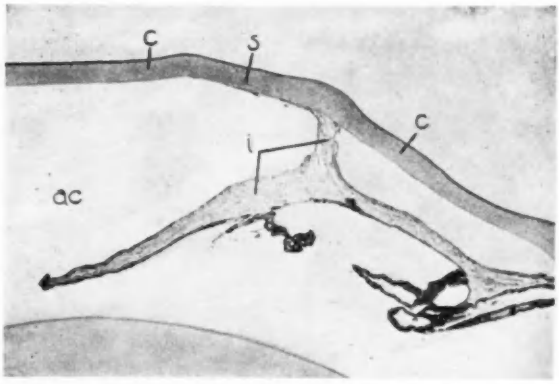

FIG. 15 — (Perera) — Section through the anterior segment of the operated right eye of rabbit No. 7 (killed four months after operation). The operative scar (*s*) is seen as an irregular mass of connective tissue in which are imbedded pigmented remains of incarcerated iris (*i*). There is an anterior adhesion of the iris lateral to the site of the scar. Serial sections of this globe showed no corneal epithelium in the scar, anterior chamber or iris. Section through the operated right eye of rabbit No. 8 (killed four months after operation) is similar to figure 15. (×30)

The rapidly growing epithelium seen in the sections of the operated right eye of rabbit No. 1 (Figs. 5-8) covered the surface of the fibrin plug, and formed a fistula into the anterior chamber. The corneal epithelium in this eye did not proliferate upon the iris, nor did it extend upon the endothelium on the posterior surface of the cornea.

In spite of the fact that an epithelial-lined passageway extended through the cornea for several days after the operation, a permanent fistula did not result in any instance in rabbits Nos. 2 to 8.

In every case, the iris became incarcerated and adherent to the corneal scar, with formation of a narrow or broad anterior synechia.

The small epithelial cyst found in the anterior portion of the corneal scar in rabbit No. 6 represents an inclusion cyst.

A survey of the serial microscopic sections allows an interpretation of the sequence of events resulting from the operation: Immediately after the operation, the corneal epithelium proliferated rapidly upon the denuded area of the corneal surface, and extended down through the keratome incision which was wedged

open by the epithelial-covered corneal flap. The down-growth of epithelium extended into and partly around the fibrin plug which formed beneath the operative incision and around the cut end of the corneal flap. The epithelium did not extend upon the iris nor upon the endothelium on the posterior surface of the cornea. Even though the corneal wound was lined by corneal epithelium, it gradually became closed off. The invading corneal epithelium and the entire implanted corneal flap were absorbed, so that by the end of the third week after the operation, no remnant was seen in the interior of the eye. The iris always became adherent and incarcerated in the scar of the keratome incision.

SUMMARY AND CONCLUSIONS

1. Epithelial invasion of the anterior chamber of the human eye following operation or injury results in "pearl" tumors of the iris, epithelial cysts of the iris, or epithelization of the anterior chamber. This group of complications is due to implantation of epithelial cells within the anterior chamber or to growth of these cells through an operative or traumatic wound.

2. The treatment of epithelial invasion of the anterior chamber has been unsatisfactory until recent years when the use of radiotherapy has given encouraging results. A case of postoperative cyst of the iris following cataract extraction successfully treated by this method is reported by the author.

3. A study of the literature on this subject, including reports of cases and experimental investigations, as well as examination of the pathological material in the collection of the Institute of Ophthalmology of the Presbyterian Hospital, New York City, shows that incarceration of iris or lens capsule following operation for removal of cataract is an important factor in epithelization of the anterior chamber. Hypotony plays a potent role, appearing to favor growth of epithelium within the anterior chamber. Operative procedures which reduce the possibility of incarceration of iris and lens capsule will reduce the incidence of postoperative growth of epithelium in the anterior chamber of the eye.

4. The author describes a study of the outcome of the experimental introduction of a flap of superficial corneal tissue into the anterior chamber of rabbits. The corneal epithelium which proliferated rapidly immediately after the operation gradually became degenerated and disappeared. The entire implanted corneal flap

became absorbed within three weeks, with formation of an anterior adhesion of the iris in all cases. Epithelization of the anterior chamber of normal rabbits could not be produced by this method.

* * *

The author wishes to thank Doctor John M. Wheeler and Doctor A. B. Reese for their kind interest, and to express his appreciation to Mr. N. Ross and Miss L. Kneiske for the technical work and microphotography, and to Mr. A. Marfaing for the photographs and lantern-slides.

REFERENCES

1. Collins, E. T.: *A large epithelial implantation cyst in a shrunken globe twenty-eight years after the receipt of injury.* Tr. Ophth. Soc. U. Kingdom. 11:133-136, 1891.
2. Mackenzie, Wm.: *A practical treatise on the diseases of the eye.* Revised by Addinell Hewson from the 4th London edition. Blanchard and Lea, Phila., pp. 672-673, 1855.
3. von Graefe, A.: *Serous cysts of the iris.* Arch. f. Ophth. 12:228-231, 1866.
   von Graefe, A.: *Ueber eine haartaltige Balggeschwulst im Innern des Auges.* Arch. f. Ophth. 3:412-418, 1857.
4. de Wecker, L.: *Ueber Cystenbildung in der Iris.* Arch. f. Augen- u. Ohrenheilk. 1:122-125, 1869.
5. Hulke, J. W.: *Report on Cases of Cysts in the Iris.* Ophth. Hosp. Rep., Lond. 6:12-27, 1869.
6. Knapp, H.: *Grosse Iriscyste, durch Operation geheilt.* Arch. f. Augen- u. Ohrenheilk. 1:175-183, 1870.
7. Rothmund, A.: *Zur Pathogenese der Iriscysten.* Klin. Monatsbl. f. Augenh. 9:397-407, 1871.
8. Monoyer, F.: *Épithélioma perle ou margaritoide de l'iris.* Ann. d'ocul. 67:249-259, 1872.
9. Feuer, N.: *Ueber serose Iriscysten.* Klin. Monatsbl. f. Augenh. 11:110-123, 1873.
10. Sattler, Hubert: *Zur Kenntniss der serosen Iriscysten.* Klin. Monatsbl. f. Augenh. 12:127-151, 1874.
11. Stolting: *Die Entstehung seroser Iriscysten.* Arch. f. Ophth. 31:99-117, 1885.
12. Collins, E. T., and Cross, F. R.: *Two cases of epithelial implantation cyst in the anterior chamber after extraction of cataract.* Tr. Ophth. Soc. U. Kingdom. 12:175-180, 1892.
    Lagrange, Felix: *Contribution à l'étude des kystes de l'iris.* Arch. d'ophth. 20:272-284, 1900.
    Goldberg, H. G.: *Implantation cyst of the anterior chamber.* Ophth. Record, Chicago. 26:212-213, 1917.
    Ahlstrom, Gustav: *Zur Kenntniss der traumatischen serosen Iriscysten.* Centralbl. f. prakt. Augenh. 27:257-262, 1903.

Williams, E.: *Report of two cases of cyst of the iris.* Tr. Am. Ophth. Soc. 3:104-110, 1880.
Frost, W. A.: *Serous cyst of iris.* Tr. Ophth. Soc. U. Kingdom. 4:58-59, 1884.
Chevallereau, and Despagnet: *Kystes de l'iris.* Ann. d'ocul. 107:280-281, 1892.
Morton, S.: *Cyst of iris.* Tr. Ophth. Soc. U. Kingdom. 13:58-60, 1893.
Eversbusch, O.: *Ein bemerkenswerther Fall von Iris-Cystenbildung.* Klin. Monatsbl. f. Augenh. 31:409-429, 1893.
Marshall, C. D.: *Implantation cysts of the iris.* Tr. Ophth. Soc. U. Kingdom. 19:54-61, 1899.
Kimpel: *Ein Fall von cystischer Epithelgeschwulst in der vorderen Kammer.* Klin. Monatsbl. f. Augenh. 38:252-256, 1900.
Fehr: *Kleine Iris-Cyste.* Centralbl. f. Augenh. 26:233, 1902.
Whitehead, A. L.: *Implantation cyst of the iris.* Tr. Ophth. Soc. U. Kingdom, 24:78-79, 1904.
Oatman, Edward L.: *Epithelial cyst formation in the iris.* Arch. Ophth. 35:309-317, 1906.
Rumbaur, W.: *Beitrage zur Klinik und Anatomie einiger seltener Tumoren des Auges und der Orbita.* Klin. Monatsbl. f. Augenh. 64: 790-801, 1920.
Roth, J. H., and Geiger, C. W.: *Posterior chamber cysts of the iris.* Am. J. Ophth., series 3. 8:870-874, 1925.
Wright, R. E.: *Cyst of the iris.* Br. J. Ophth. 9:454-456, 1925.
Paderstein: *Traumatische serose Iriszyste.* Ztschr. f. Augenh. 73: 292, 1931.
Bonnet, P., and Paufique, L.: *Kystes traumatiques de l'iris* Arch. d'ophth. 51:5-16, 1934.
Tooker, Charles W.: *Epithelial cyst in the posterior chamber.* Am. J. Ophth., series 3. 17:41-47, 1934.
T'and, Y. T.: *Pigmented epithelial cyst of the iris.* Chinese M. J. 48: 905-909, 1934. (Abstracted in Am. J. Ophth., series 3. 18:282, 1935).
Bracci-Torsi, H.: *Contributo clinico ed istologico alle cisti dell'iride.* Ann. di. ottal. e clin. ocul. 63:65-73, 1935.

13. Rothmund, A.: *Ueber Cysten der Regenbogenhaut.* Klin. Monatsbl. f. Augenh. 10:189-223, 1872.
14. de Wecker, L.: *Ueber cystoide Entartung der Iris.* Klin. Monatsbl. f. Augenh. 11:229-236, 1873.
15. Hosch, F.: *Beitrag zur Lehre von den serosen Iriscysten.* Klin. Monatsbl. f. Augenh. 12:119-127, 1874.
16. Greef, R.: *Zur Kenntniss der intraocularen Cysten.* Arch. f. Augenh., Wiesb. 25:395-416, 1892. (Translated by Holden, W. A.: Arch. Ophth. 22:481-492, 1893).
17. Collins, E. T.: *On the pathology of intraocular cysts.* Ophth. Hosp. Rep., Lond. 13:41-77, 1893.
18. Urmetzer, J.: *Ueber Epitheleinwanderung und Entwickelung von Epithelcysten im Auge.* Arch. f. Ophth. 68:494-533, 1908.
19. Ginsberg, S.: *Traumatische Iriszysten.* Handb. d. spez. path. Anat. u. Histol., Julius Springer, Berlin. Edited by F. Henke and O. Lubarsch. 11:546-550, 1928.
20. Villard, H., and Dejean, Ch.: *Les Kystes de l'iris.* Arch. d'ophth. 50:91-121; 194-211; 272-287; 1933.
21. Guaita, M.: *Prolifération de l'épithélium cornéen sur l'iris et le champ pupillaire après l'extraction de la cataracte.* Arch. d'ophth. 13:507-508, 1893.

22. Meller, J.: *Ueber Epitheleinsenkung und Cystenbildung im Auge.* Arch. f. Ophth. 52:436-445, 1901.
23. Elschnig, A.: *Epithelauskleidung der Vorder-und Hinterkammer als Ursache von Glaukom nach Staroperation.* Klin. Monatsbl. f. Augenh. 41:part 1, 247-253, 1903.
24. Ginsberg, S., and Hirschberg, J.: *Ein Fall von Vorderkammer-Cyste.* Centralbl. f. prakt. Augenh. 33:225-235, 1909.
25. Collins, E. T.: *Discussion on postoperative complications of cataract extractions.* Tr. Ophth. Soc. U. Kingdom. 34:18-44, 1914.
26. Hagen, S.: *Corneoscleral cyst with epithelial infiltration in the anterior chamber and secondary glaucoma.* Norsk. mag. f. lægevidensk. 77:1567-1577, 1916.
27. Berg, F.: *Ein Fall von operierter Vorderkammerzyste mit Flachenausbreitung des Zystenepithels an der Linse und zeitweiser Rückbildung der Epithelproliferation.* Klin. Monatsbl. f. Augenh. 65:879-882, 1920.
28. Strader, G. L.: *Cyst in anterior chamber.* Am. J. Ophth., series 3. 3:448, 1920.
29. Ballacco: *Invasione epiteliale papillomatosa della camera anteriore.* Atti del Congresso d'Oftalmologia, Rome. 148-150, 1926.
30. Salus, R.: *Geheilte Epithelauskleidung der Vorderkammer.* Klin. Monatsbl. f. Augenh. 78:368-371, 1927.
31. Levine, J.: *Growth of corneal epithelium into anterior chamber.* Am. J. Ophth., series 3. 16:796-798, 1933.
32. Marcove, M. E.: *Implantation cyst of the anterior chamber following injury to the cornea.* Am. J. Ophth., series 3. 17:533, 1934.
33. Critchett, G.: *Hour-glass cyst of iris following a wound.* Ophth. Hosp. Rep., Lond. 9:53-54, 1879.
34. Axenfeld, Th.: *Weitere Erfahrungen ueber intraokulare Strahlentherapie.* Ber. u. d. 41 Versamml. d. Ophth. Gesellsch., Heidelberg. 324-327, 1918.
25. Jendralski, F.: *Strahlentherapie der Iriszysten.* Klin. Monatsbl. f. Augenh. 68:175-179, 1922.
36. Handmann, M.: *Vollstandige Ruckbildung einer traumatischen Iriszyste nach Rontgenbestrahlung.* Klin. Monatsbl. f. Augenh. 72:111-116, 1924.
Handmann, M.: *Vorstellung eines Kranken mit traumatischer Iriszyste, die durch Rontgenbestrahlung beseitigt wurde.* Klin. Monatsbl. f. Augenh. 74:218, 1925.
37. Custodis, E.: *Beitrag zur Pathologie und Therapie der Iris- und Iriskammer-Zysten.* Klin. Monatsbl. f. Augenh. 90:361-368, 1933.
38. Vail, D.: *Epithelial downgrowth into the anterior chamber following cataract extraction arrested by radium treatment.* Tr. Am. Ophth. Soc. 33:306-325, 1935.
39. Gradle, H. S.: *Conjunctival drain of the anterior chamber.* Tr. Sect. Ophth., A.M.A. 329-340, 1927.
40. Fuchs, E.: *On keratitis* (The Bowman Lecture). Tr. Ophth. Soc. U. Kingdom. 22:15-34, 1902.
41. Weinstein, A.: *An experimental study of the healing of perforating incised wounds of the cornea.* Arch. Ophth. 34:32-61, 1905.
42. van Dooremaal, J. C.: *Die Entwickelung der in fremden Grund versetzten lebenden Gewebe.* Arch. f. Ophth. 19:359-373, 1873.
43. Goldzieher, Wilhelm: *Ueber Implantationen in die vordere Augenkammer.* Arch. f. exper. Path. u. Pharmakol. 2:387-404, 1874.
44. Schweninger, Ernst: *Ueber Transplantation und Implantation von Haaren.* Ztschr. f. Biol. 11:341-380, 1875.

45. Hosch, F.: *Experimentelle Studien ueber Iriscysten.* Virchow's Archiv. 99:448-459, 1885.
46. Corrado, M.: *Glaucoma secondario a penetrazione e proliferazione di epitelio in C. A. in occhio operato di cataratta.* Ann. di ottal. e clin. occul. 59:706-717, 1931.
47. Accardi, V., and Rossi, E. G.: *Implantation of tissues into the Anterior Chamber.* Rassegna Ital. d'ottal. 5:3-54, 1936.
48. Federici, Ermanno: *I raggi Roentgen nell'epitelizzazione della camera anteriore e nella ritardata guarigione delle ferite operatorie nei cataratosi.* Bollettino d'Oculistica. 16:112-125, 1937.

DISCUSSION

Dr. F. Bruce Fralick, Ann Arbor, Mich.: The instructive paper which Dr. Perera has presented to us today abstracts our present knowledge of the subject of the presence of epithelium in the anterior chamber of the eye. Undoubtedly this dreaded complication of intraocular operations or perforating ocular injuries is of very infrequent occurrence, as illustrated by the fact that this condition has been observed but twice in the past decade in the Ophthalmological Clinic of the University of Michigan. The two cases seen by us followed perforating injuries, one through the center of the cornea resulting in an epithelial cyst of the anterior chamber and the other an hour glass epithelial cyst lining the anterior and posterior chambers which communicated through the limbus with a large subconjunctival epithelial lined cyst. In the latter case the injury was apparently to the conjunctiva, together with a subconjunctival perforation at the limbus into the anterior chamber. Both eyes were glaucomatous when seen. In both instances serial sections revealed evidence of iris tissue incarcerated in the perforation as found in the author's cases.

That foreign tissue in the anterior chamber does have a strong tendency to absorb was observed by Elschnig, and this may account for the fact that epithelization of the anterior chamber does not occur more often. At one time I was fortunate in being able to observe at frequent intervals a trephined eye in which the scleral button had been lost in the anterior chamber. The button became smaller and smaller each week and at the end of a month was entirely absorbed.

It is encouraging to hear of another favorable therapeutic result through the use of X-ray therapy, since most conscientious X-ray therapists will not admit of a selective affinity of X-rays for normal surface epithelium. If no such selective action on the part of X-rays exists, the question must be asked as to how X-rays could destroy surface epithelium growing in the anterior chamber and yet not the same epithelium on the anterior corneal surface where it is closer to the source of radiation and thus receiving a more intense dosage. It might be that the epithelium in the anterior chamber is growing under such unfavorable circumstances that it is vulnerable to a dosage of X-rays which would have no harmful effects on the same tissue when growing in its normal environment.

The quality of the X-ray used by the author is that which is considered as "deep therapy." In epithelial cysts of the anterior chamber we are not concerned with deep penetration but rather with the superficial therapeutic effects; so that the filtration and K.V.P. used could be considerably decreased and yet get the same ionization in the anterior chamber of the eye. This suggestion is offered not as a criticism of the author's X-ray technic, since a brilliant result has been obtained, but merely to point out that the same ionization or destruction of the epithelial cells in the anterior chamber should be obtained without the use of X-rays which have such depth of penetration. If a low penetrating beam of X-rays, generated at low voltages, were used instead of "deep therapy" it might be an important factor in preventing cataract formation or vascular degeneration in the retina or choroid in other cases.

Dr. Derrick Vail, Cincinnati: I should like to draw your attention to some experimental work along these same lines, which, because it is in the Japanese language, has probably escaped your attention. I refer to the excellent researches of Dr. Z. Suzuki* of Kyoto. A Japanese colleague of mine, Dr. Shiro Tashiro of Cincinnati, very kindly translated the important features of these articles for me.

Suzuki experimentally produced epithelial lined cysts of the anterior chamber and iris. He found that cysts could only be produced by introducing (1) a strip of conjunctiva into the anterior chamber attached to a stalk. This was most frequently successful. (2) Isolated strip of whole conjunctiva. This succeeded in producing an iris cyst in only 6 out of 19 attempts; and (3) epithelium from the conjunctiva only. This invariably failed. In other words, it seems that it is necessary for epithelium to proliferate within the anterior chamber that it have attached to it connective tissue.

Having successfully produced cysts experimentally he subjected them to x-rays and reached the following conclusions:

1. By producing experimentally iris cysts in rabbits and treating them with 1 H.E.D. once, or 3 H.E.D. in 3 doses, he found the cysts to shrink within 3-4 months. There was almost complete disappearance after 9-10 months. Histologically speaking, epithelial cells of the anterior walls of the cyst underwent a great change, and he often observed their complete disappearance. No such change was found on the posterior wall, which is directly connected with the iris, and favorably situated for nutritional advantage.

2. In the cases where transplantation was made for the purpose of producing the cysts, if x-ray treatment was given (1-2 H.E.D.) before the

* Suzuki, J.:

1. *Experimental studies on the development of Traumatic Serous iris cysts.* Acta. Soc. Oph. Janan, Supplement, Oct., 1930, vol. 34, Fasiculus X.
2. *The Therapeutic Effect of Roentgen Ray on Traumatic Serous Iris Cysts.* Acta. Soc. Oph. Janan. pp. 2-4, 14-15, 1934.

appearance of the cysts, no epithelial changes were found, due probably to the good nutrition brought through the piece of conjunctiva. Thus, he concluded that x-ray does not prevent cyst formation.

DR. W. E. FRY, Philadelphia: The problem of the ingrowth and proliferation of corneal epithelium into the anterior chamber with the formation of an anterior chamber cyst is not a new one. It is a condition that has been clinically recognized for perhaps half a century. Until relatively recently the occurrence of this condition has been disastrous as far as its effect upon the eye is concerned. Recently the reports of irradiation therapy are exceedingly favorable, and the case that Dr. Perera reports here at this meeting illustrates this very vividly. I think Dr. Perera has developed a very interesting experimental method to study this condition. Although his results are essentially negative, I believe they are nevertheless important.

One obvious conclusion which one may draw from this is that corneal epithelium implanted in the eye of a normal, healthy rabbit is rapidly absorbed and destroyed, and in order to obtain growth and continued proliferation of epithelium into the anterior chamber other factors are necessary, such as trauma, inflammation, or some degenerative condition.

In slides of this condition, of which I have been able to examine some half dozen which are in the collection of the Wills Eye Hospital, there is either absence of or marked degenerative changes in the corneal endothelium. In one such slide the endothelium is absent even at places where epithelial tissue does not cover the posterior corneal surface.

Previous experimenters have reported fistulization and continued hypotonia as important predisposing causes of proliferation of epithelium. It does not seem to me that the continued hypotonia is a primary cause, but rather that this continued hypotonia causes other damages in the eye which permit epithelium to continue to grow.

Experimenters working in another field, particularly Markee, have reported, for instance, endometrial tissue implanted in the anterior chamber of a healthy rabbit will have exactly the same measured area at the end of eighteen months as it had at the time of beginning the experiment. He does not mention glaucoma as among the complications in his experiments.

There is another phase of this subject that has been of interest to me, and that is illustrated by sections from an eye, which I should like to present to you. Through the courtesy of Dr. DeLong I was able to have lantern slides made of this. It is a case which was in the Wills Eye Hospital. On this slide corneal epithelium extends over the anterior surface of the iris, around the pupillary border, over the capsular remains, and over strands extending into the vitreous chamber. At several places cystic degenerative changes have occurred in this epithelium.

The reason why this has been particularly interesting to me is this: That there might be some relationship between this type of change and the small, transparent globular bodies which at times are found in the pupillary area after cataract extraction. With Dr. Cowan I was able to report some such cases before this section last year. In most of such cases there had

been an extracapsular extraction. In one of our cases an intracapsular extraction was done, and in such a case I do not believe that the reason assigned by us at that time would account for these globules. The reason given at that time was proliferation and degeneration of capsular epithelium. It seems to me that a possible cause is implantation of corneal epithelium with its resulting cyst formation and degeneration. We have seen such cases in which the cysts change rapidly and frequently disappear. This would correspond quite well to the experimental experience which Dr. Perera has shown us this afternoon.

[Dr. Fry showed three slides illustrating growth of epithelium over the surface of the iris, around the pupillary margin, into the vitreous. Cyst formation by the ingrowth of corneal epithelium was demonstrated.]

DR. CHARLES A. PERERA, closing: I wish to thank Dr. Fralick and Dr. Fry for their very interesting discussion, and to express my appreciation to Dr. Vail for his presentation of some work in Japan, which I had not yet encountered.

REFRACTIVE ERRORS IN THE SAME EYES WHILE UNDER THE INFLUENCE OF HOMATROPINE, SCOPOLAMINE AND ATROPINE

LOUIS BOTHMAN, M.D.*
CHICAGO

A COMPARISON of the changes in refraction of the same eyes while under the influence of homatropine and atropine appeared in 1932.[1] A similar study of the refractive changes of the same eyes while under the cycloplegic effect of scopolamine and atropine was published in 1937.[2] Because of the considerable differences found in these series, I decided to compare the findings in the same eyes while under the effect of all the drugs most commonly used in doing retinoscopy. This is a report on the analysis of 200 such eyes. Because the changes in the previous series were greater in younger eyes, all patients in this series are under 40 years of age. The material is drawn from the University of Chicago Clinics and private practice.

The material presented consists of 100 cases (200 eyes). It is divided into Group A, patients under 20 years of age, which contains 48 cases, 35 male and 13 female, and Group B, patients over 20 years old, which contains 52 cases of whom 19 are male and 33 female. For the series there were 54 males and 46 females. In Group A, 14 patients were less than 10 years and 34 were more than 10 years old. In Group B, 31 were under 30 years and 21 more than 30 years of age.

The refractive errors varied from emmetropia to minus 10.00 D (one eye) and plus 7.00 D (2 eyes). The average error under 20 years of age was 2.23 D in myopia and 1.42 D in hyperopia. In Group B, the average error was 2.00 D in myopia and 1.75 D in hyperopia. For the entire series the average error was 2.10 D in myopia and 1.75 D in hyperopia.

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TECHNIQUE

All of the eyes were examined by retinoscopy at 1 meter distance using a concave mirror.

Homatropine hydrobromide in a 2% aqueous solution was instilled into the conjunctival sacs. Six drops were used at 10 minute intervals and the retinoscopy was done 10 minutes after the final drop.

Scopolamine hydrobromide was used in ½% aqueous solution. Two drops were used at half hour intervals and the test made 15 minutes after the last drop.

Atropine sulphate in a 1% aqueous solution was used before retiring the first night and four times daily for three days and in the morning of the day of the examination. In all, 14 drops of atropine were used.

The percentage of toxic reactions due to scopolamine has dropped from 8 and 4% in previous reports to less than 3% in the present series. This is probably due to insistence on pressure over the tear sacs for 10 minutes after using the drops and care on the part of the druggist in making a ½% solution.

DATA

The following tabulation indicates the types of changes found with the three cycloplegics.

In Group A —

In 2 eyes a myopic error under homatropine and scopolamine became hyperopic under atropine.

In 1 eye myopia under homatropine and scopolamine changed to mixed astigmatism under atropine.

In 1 eye myopia under homatropine and scopolamine became emmetropia under atropine.

In 3 eyes mixed astigmatism under homatropine and scopolamine, became hyperopia under atropine.

In 1 eye, mixed astigmatism under homatropine became hyperopia under scopolamine and atropine.

In Group B —

In 2 eyes, myopia under homatropine and scopolamine became mixed astigmatism under atropine.

In 2 eyes, myopia under homatropine became mixed astigmatism under scopolamine and atropine.

In 3 eyes, myopia under homatropine changed to hyperopia under scopolamine and atropine.

In 8 eyes mixed astigmatism under homatropine and scopolamine became hyperopia under atropine.

In 3 eyes mixed astigmatism with homatropine became hyperopia with scopolamine and atropine.

In 1 eye emmetropia under homatropine showed hyperopia with scopolamine and atropine.

In 2 eyes emmetropia with homatropine changed to mixed astigmatism under scopolamine but were emmetropic with atropine.

In 14.5% of the whole series, the type of refractive error changed with more complete cycloplegia.

DIFFERENCES IN DIOPTERS

The average difference was for hyperopia 0.21 diopters more per meridian in patients under 20 years, 0.14 D in patients more than 20 years of age and 0.17 D in all hyperopic eyes with homatropine and scopolamine.

The difference for group A was 0.33 diopters higher, group B 0.14 D higher and for all hyperopic meridians 0.23 D higher with atropine than with scopolamine.

With scopolamine the myopic meridians in group A required an average of 0.16 D less correction, group B 0.19 D less and all myopic meridians 0.18 D less than with homatropine.

Under atropine group A averaged 0.25 D less; B, 0.17 D less and all myopic meridians 0.21 D less than with scopolamine.

The difference between homatropine and scopolamine in the mixed astigmatism meridians averaged for group A, 0.16 D, group B, 0.17 D and for all 0.15 D.

With scopolamine and atropine, the difference was for group A 0.10 D; B, 0.19 D; and for all, 0.15 D.

For all meridians, 200, the average difference between homatropine and scopolamine was 0.168 D and between scopolamine and atropine 0.159 D.

Comparison between homatropine and atropine is omitted as a much larger series (1,260 eyes) has been previously reported.[1]

Table II indicates the type of refraction errors with the changes found in each eye.

HYPEROPIA

48.2% of the hyperopic eyes required stronger sphere under scopolamine than under homatropine while 55.3% were higher under atropine than with scopolamine.

The combination of the same sphere with a stronger cylinder occurred in 20.5% of the eyes under scopolamine and in 13% under atropine.

TABLE I — *Changes in Meridians of Refraction*

		No. of Eyes	Meridians	>	No Change	<	Difference +0.25	+0.50	+0.75	+1.00	Average in Diopters	Astigmatism With Rule Hom.	Scop.	Atr.	< C	> C	1 C
Hyperopia	Hom. & Scop.																
	A	53	106	74	23	9	40	18	13	3	0.21	33/42	36/48	38/50	11	17	22
	B	53	106	43	53	10	25	15	2	1	0.14	30/42	37/52	34/49	7	28	20
	Total	106	212	117	76	19	65	33	15	4	0.17	63/84	73/100	72/99	18	45	42
	Scop. & Atr.																
	A	53	106	79	20	7	43	24	9	3	0.33				10	19	20
	B	53	106	49	42	15	30	15	3	1	0.14				14	20	33
	Total	106	212	128	62	22	73	39	12	4	0.23				24	39	43
Myopia	Hom. & Scop.																
	A	34	68	35	29	4	21	13	1		0.16	22/28	21/29	24/33	1	11	20
	B	34	68	43	20	5	31	9	3		0.19	17/29	16/29	18/30	3	13	12
	Total	68	136	78	49	9	52	22	4		0.18	39/57	37/58	42/63	4	24	22
	Scop. & Atr.																
	A	34	68	49	18	1	27	15	7		0.25				11	12	12
	B	34	68	34	26	8	23	9		2	0.17				6	8	16
	Total	68	136	83	44	9	50	24	7	2	0.21				17	20	28
Mixed Astigmatism	Hom. & Scop.																
	A	9	18	12	6		6	4		2	0.16	7/7	7/7	7/7	4	2	2
	B	17	34	17	17		10	5	1	1	0.17	12/12	12/12	12/12	2	4	8
	Total	26	52	29	23		16	9	1	3	0.15	19/19	19/19	19/19	6	6	10
	Scop. & Atr.																
	A	9	18	13	5		12	1			0.10				3	2	3
	B	17	34	20	12	2	9	10		1	0.19				7	2	5
	Total	26	52	33	17	2	21	11		1	0.15				10	4	8
Hom. & Scop.		200	400	224	148	28	133	64	20	7	0.168				28	75	74
Scop. & Atr.		200	400	244	123	33	144	74	19	7	0.159	121/160	129/177	133/181	51	63	79

A = Patients under 20
B = Patients 20 to 40 years

> = More
< = Less

1 = Same
S = Sphere

C = Cylinder

TABLE II — *Changes in Refraction of Each Eye*

		No. of Eyes	> S	< S	1 S	>S <C	>S >C	>S 1C	<S <C	<S >C	<S 1C	1S >C	1S <C	1S 1C	1 Axis	< 15° Diff.	> 15° Diff.	S to Ast.	Ast. to S
HYPEROPIA H&S	A	53			3	9	8	18	1	3	2	6		3	19	17	4	7	3
	B	54	2		1	4	7	5	1	3	1	16	1	13	20	21	4	8	2
	TOTAL	107	2		4	13	15	23	2	6	3	22	1	16	39	38	8	15	5
HYPEROPIA S&A	A	53	1			8	15	15	1	3		7		3	22	19	5	3	
	B	54	1		3	12	6	4	0	5	1	7	2	13	24	19	5	3	5
	TOTAL	107	2		3	20	21	19	1	8	1	14	2	16	46	38	10	6	5
MYOPIA H&S	A	34		1	1				2	9	10	2		9	17	10	1	4	2
	B	34		2		2			2	13	5	5	1	4	17	10	2	3	1
	TOTAL	68		3	1	2			4	22	15	7	1	13	34	20	3	7	3
MYOPIA S&A	A	34				2			5	11	7		5	4	18	16	3	1	
	B	34				2	1	2	3	6	6	2	5	7	12	9	5	3	
	TOTAL	68				4	1	2	8	17	13	2	10	11	30	25	8	4	
MIXED ASTIGMATISM H&S	A	9							1		1	2	2	3	5				
	B	16					2	1	1	3	3	1		5	8	8	2		
	TOTAL	25					2	1	2	3	4	3	2	8	13	8	2		
MIXED ASTIGMATISM S&A	A	9				1	1				6	1			5	5			
	B	16				3		1	2	2	2	1	2	3	5	10			
	TOTAL	25				4	1	1	2	2	8	2	2	3	10	15			
GRAND TOTAL	Hom. & Scop.	200	2	3	5	15	17	24	8	31	22	32	4	37	86	66	13	22	8
	Scop. & Atr.	200	2		3	28	23	22	11	27	22	18	14	30	86	78	18	10	5
HYPEROPIA IN %	Hom. & Scop.		1.8	0	3.6	12.8	14.0	21.4	1.8	5.4	2.8	20.5	0.9	14.6	36.4	35.5	7.4	14.0	4.5
	Scop. & Atr.		1.8	0	2.8	18.0	19.6	17.7	0.9	7.2	0.9	13.0	1.8	14.6	42.9	35.5	18.0	54.0	4.5
MYOPIA IN %	Hom. & Scop.		0	4	1.4	2.8	0	0	5.6	32.3	22.0	10.2	1.4	19.1	50.0	28.0	4.0	10.2	4.0
	Scop. & Atr.		0	0	0	5.6	1.4	2.8	11.3	25.0	19.1	2.8	14.7	16.1	40.0	14.0	11.3	5.6	0

A = Patients under 20
B = Patients 20 to 40
S = Sphere

C = Cylinder
> = More
< = Less

1 = Same
Diff. = Difference
Ast. = Astigmatism

H&S = Homatropine and Scopolamine
S&A = Scopolamine and Atropine

Of the 11 eyes which required weaker spheres with scopolamine than with homatropine, 6 had stronger cylinders.

In the 10 eyes with less sphere under atropine than under scopolamine, 8 required higher cylinders.

MYOPIA

64.5% of the myopic eyes required weaker spheres under scopolamine than were found under homatropine and 32.3% were combined with weaker cylinders.

55.4% of the myopic eyes had weaker spheres under atropine than under scopolamine, 25% being combined with weaker cylinders.

With homatropine and scopolamine 30.7% required the same strength sphere and 19.1% a like strength cylinder.

With scopolamine and atropine 33.6% had the same sphere; there were 14.7% with less and 16.1% with the same cylinder.

Under scopolamine 2 eyes required more sphere but less cylinder than under homatropine.

Of the 7 eyes requiring more sphere under atropine than under scopolamine 4 had less and 2 the same strength cylinder.

Table I indicates the changes of refraction of each meridian.

HYPEROPIA

31.5% of all meridians in patients under 20 years of age had 0.50 or more diopters more hyperopia under scopolamine than under homatropine. In the group over 20 years of age this was true in 16.9% of all meridians. 24.5% of all hyperopic meridians were 0.50 or more diopters stronger under scopolamine than under homatropine.

33.9% of the younger group and 17.9% of patients over 20 years of age required more than 0.50 diopters increase of each hyperopic meridian under atropine than was found with scopolamine. 25.9% of all hyperopic meridians varied more than 0.50 diopters with atropine and scopolamine.

MYOPIA

20% of the younger and 17.6% of the older group or 19.1% of all myopic meridians were 0.50 or more diopters weaker under scopolamine than under homatropine.

32.3% of the younger, 16.1% of the older or 24.3% of all myopic meridians were 0.50 or more diopters weaker under atropine than with scopolamine.

MIXED ASTIGMATISM

33.3% of Group A, 20.5% of group B, or 25% of all mixed astigmatism meridians varied 0.50 or more diopters with scopolamine and homatropine.

5.5% of group A, 32.2% of group B, or 23% of all mixed astigmatism meridians varied more than 0.50 diopters with atropine and scopolamine.

ASTIGMATISM

14% of the eyes with plain spheres under homatropine had astigmatism with scopolamine and 5.4% with only spherical corrections under scopolamine required cylinders under atropine.

4.5% of the eyes with astigmatism while under homatropine required no cylinder when under scopolamine and the same percentage with cylinders under scopolamine became spherical corrections only under atropine.

The axis of the astigmatism remained the same in 50% with both homatropine and scopolamine while 40% were the same with both scopolamine and atropine.

There were 28% of the astigmatic eyes under homatropine which had less, and 4% with more than 15° difference in the axis under scopolamine.

14% had less and 11.3% more than 15° difference in the axis with scopolamine and atropine.

The axes in the mixed astigmatism eyes were almost the same with all three drugs.

Astigmatism was with the rule 121 times in 160 eyes under homatropine; 129 times in 177 with scopolamine and 133 times in 181 eyes under atropine.

SUMMARY AND CONCLUSIONS

A series of the same 200 eyes in patients under 40 years of age has been refracted while under the cycloplegic effect of homatropine, scopolamine and atropine and the findings compared.

While the average difference in diopters seems very small and is less than in larger series previously reported, the fact that in 14.5% of the series the type of refractive errors changed, that 1.75% of all meridians varied more than 1 diopter, that 24.5% of hyperopic meridians with homatropine and 25.9% with scopolamine varied more than 0.50 D, that 19.1% of all myopic meridians with homatropine and 24.3% under scopolamine were more

than 0.50 D weaker, that 25% of the mixed astigmatism meridians under homatropine and 23% under scopolamine varied 0.50 or more diopters, that 11.3% of the axis of the astigmatism under scopolamine changes more than 15° and in a few instances even 90° is very significant.

Scopolamine is a more complete cycloplegic than homatropine, while atropine gives more complete cycloplegia than either homatropine or scopolamine.

REFERENCES

1. L. Bothman: Arch. of Ophth. 7:389, 1932.
2. L. Bothman: Amer. Jour. of Ophth. 20:822, 1937.
3. L. Bothman: Ill. Med. Jour. 71:229, 1937.

DISCUSSION

Dr. Robert J. Masters, Indianapolis: Fifteen years ago, at the very beginning of our work at the Graduate School of Medicine of the University of Pennsylvania, our class in ophthalmology heard its first lecture on refraction, which started with the following exhortation:

"Work hard at the job of learning to refract well. Work diligently from the very beginning to constantly improve yourselves in the testing of eyes for correcting lenses. Eighty per cent of your work will be refraction work. No matter how long you may work or how successful you may become, your refraction work will always be the back-bone of your practice."

All of us have found this statement to be very true, without need of modification as time has passed. We have, therefore, tried to improve our methods as we have gone along by making changes that have been suggested by our own experience and that of our colleagues. We may differ from one another somewhat in many minor details of our testing technique, but most of us are agreed upon one thing, namely, that the use of a cycloplegic is absolutely essential to the accurate measurement of the refraction of eyes under forty-five years of age.

In many of the smaller cities the oculists meet with a great deal of opposition to the use of "drops." In these cities the local optometrists are a serious competitive factor, and frequently the oculists feel that they must forego the use of a cycloplegic to avoid an argument or the loss of a patient. To these gentlemen it should be said that it is wrong to test the eyes of people under forty-five years of age without a cycloplegic, unless for some good reason the use of a cycloplegic is contraindicated. Furthermore, it is a mistake to fail at any time to draw a sharp line of distinction between the eye physician, privileged to use drugs when needed, and the optometrist who lacks that privilege. The public can be educated to understand this distinction, even if it fails to comprehend some others.

There are some ophthalmologists of experience who are using cycloplegics *occasionally* instead of *consistently*, with the convenience of the patient given as the reason for testing without "drops." There are patients whose eyes would be jeopardized by prolonged dilatation of the pupils. But when the inconvenience of temporarily blurred vision is all that holds the oculist and patient back, they too should be reminded that it is wrong to test the eyes without a cycloplegic, unless for some good reason its use is contraindicated.

With most of us agreed, then, that the test under cycloplegia is the foundation upon which a satisfactory refraction test is built, we are still faced with the problem of what cycloplegic we should use, and by what method of its use we may obtain the best results. Although we discount the patient's convenience to the extent of using "drops," we still wish to shorten as much as possible the period of his inconvenience from blurred vision. There are some of us who have wondered whether the efficiency of a cycloplegic is indicated by the duration of its action. Is atropine productive of more profound cycloplegia than that obtained with scopolamine and homatropine when they are at the peak of their cycloplegic effect? Are we quite sure when this peak of cycloplegic effect occurs, and how long it lasts? Of course, I believe we are all agreed that no cycloplegia gives us complete cycloplegia, no matter what one we use or how we use it. What we want is a clinical, working cycloplegia. In addition to these questions, we should ask ourselves if we are using each of the three commonly employed cycloplegics in such a way as to get the most good from them.

We are all indebted to the essayist for his careful, extensive and laborious studies reported in his present and two previous communications. These studies are unusual. Few of us have had the experience of studying the cycloplegic effect of atropine, scopolamine and homatropine, respectively, upon the same eyes within a short period of time. We have, therefore, studied Dr. Bothman's reports with great interest, and have been led by proof to the conclusion that atropine, the cycloplegic of longest duration, is also the most effective.

Personal comment upon these studies must be of the most elementary type, since personal knowledge is still, after fifteen years of work, most elementary. Personal experience with scopolamine is unworthy of comment, as it is relatively very small. Several early observations of its toxic effect in the practice of others (even when the canaliculi were carefully compressed) caused an antipathy to it which has never been overcome. This attitude may be wrong, but scopolamine has been little used by me for the testing of refraction. Atropine is, of course, the cycloplegic which must be used in the very young. The age at which it may give way to one of the other drugs is always a point for argument. Our essayist has said that it should be used up to sixteen years of age. I only occasionally use it after twelve years of age, changing then to *homatropine*, instead of scopolamine.

Homatropine should not be used in a solution as weak as 2%. In this strength it has seldom been an effective cycloplegic in my experience. For satisfactory results a 4% solution should be employed, with or without the addition of cocaine to make a 2% solution of the latter. Five instillations at ten to fifteen minute intervals, with retinoscopy begun from 70 to 80 minutes after the first instillation, have given cycloplegia that has been

consistently satisfactory during seven years of experience with it. The drops are always instilled by my brother or myself. I have mentioned this only to drive the point home that it is better for the physician to instill the drops himself than to have an office assistant do it, and we all know that it is more satisfactory to do it ourselves than to have the parents at home instill the drops for us. The solution is strong enough that it tends to smart somewhat. Several years ago Dr. Edward Jackson taught me that warm solutions do note tend to smart in the eye as much as cold ones. The solution of homatropine is, therefore, always warmed before its instillation, and there is no serious complaint about it, even from the younger or more apprehensive patients.

The cycloplegic test has been referred to earlier as the foundation upon which a satisfactory refraction test is built. No discussion of cycloplegics should be concluded, however, without expressing the thought that the precycloplegic manifest test supplies a useful preliminary excavation, and the post-cycloplegic test furnishes an indispensable superstructure. Without at least the post-cycloplegic superstructure, the cycloplegic foundation all alone makes a very incomplete picture.

Dr. John F. Gipner, Rochester, N. Y.: In 1864, Donders wrote, "A comparative examination of a number of substances and preparations has shown that the most useful mydriatics are to be found among the Solaneæ, and that of these the Atropa Belladonna is, for various reasons, to be preferred to all others, even to the Datura Stramonium and the Hyoscyamus niger."

Today Dr. Bothman again calls our attention to the fact that atropine still holds first place as the complete cycloplegic when he compares the refractive errors in the same eyes while under the influence of homatropine, scopolamine and atropine.

Dr. Bothman is obviously in favor of atropine since he uses fourteen drops of 1% atropine solution within three and a half days and only the generally accepted minimal doses for the weaker drugs. As one who has found homatropine cycloplegia usually satisfactory, I wish that he had made, or would in the future make, a better case for the two weaker cycloplegics, allowing them a better chance to approach complete cycloplegia by varying the dose or strength of the solutions as is found necessary.

Certainly the almost universal use of the weaker cycloplegics reflects the demand of the public for the least prolonged disability to the accommodation which is commensurate with reasonable accuracy in measuring refractive errors.

Whether or not one will use atropine in routine refraction work will largely depend upon one's philosophy of the art and science of refraction.

If one proceeds with the concept that the full correction of the refractive errors as found under complete cycloplegia is necessary for the comfort of the patient, and that one can only consider a scientific reduction of the patient's refractive error to the emmetropic state with glasses, then one will use atropine and atropine alone in examining all refraction cases.

However, if one proceeds with another philosophy of the art and science of refraction and chooses to be less the scientist but more the artist in examining his patients, and if one respects hypertrophy of the ciliary muscle as physiological and worthy of keeping as long as symptoms of accommodative strain are controlled, one will then give more consideration to the control of the accommodation in refraction with milder cycloplegics and will also master the art of manifest refraction.

Dr. Bothman's paper is a challenge for those of us who use homatropine to improve our refraction methods with this drug. Improvements can be made up to the pharmacological limits of action of the drug. These improvements consist in the proper instillation of the homatropine solution which has been freshly prepared after the manner described years ago by Dr. Edward Jackson; the use of a stronger than 2% solution, if necessary, such as a 3% solution without cocaine; performing retinoscopy with the patient fixing the gaze on a small light at least twenty feet away, so that the examiner has the benefit of the patient's voluntary relaxation of accommodation during distance fixation as well as the cycloplegic action of homatropine.

To these measures designed to get the best results from a known incomplete cycloplegic is added the measuring of the range of the residual accommodation with a Prince rule while a plus three lens has been placed over the lens combination which corrects the vision for distance. If the residual accommodation is 1½ to 2½ or more diopters, one can attempt further relaxation with more homatropine or use atropine and re-examine later.

When homatropine is employed, as I have outlined, a satisfactory retinoscopy and refraction will be secured in most cases.

Incomplete cycloplegia may produce a bizarre action on the ciliary muscle. Dr. Bothman found two eyes emmetropic with homatropine which showed mixed astigmatism under scopolamine and which were emmetropic again under atropine. Obviously, the scopolamine cycloplegia was inadequate while the homatropine cycloplegia must have been quite complete. Could not this insufficiency of cycloplegia with scopolamine have been discovered by measuring the residual range of accommodation? Would it not have been relieved by another drop or two of scopolamine? Should we not expect varying susceptibilities to cycloplegics in our patients and not rely on the routine 2 drops of scopolamine to accomplish all that we want in all cases?

If one accepts the second mentioned philosophy of the art and science of refraction and uses homatropine cycloplegia, one is not too much concerned with the failure to uncover the total hyperopia or hyperopic astigmatism, or the decrease in myopia or myopic astigmatism which might be elicited under atropine. Indeed in the case of myopia, a very slight over-correction which might be ordered from homatropine cycloplegia findings may actually be better for the control of progressing myopia than the full correction found under atropine. This I have discovered since Dr. Edward Jackson called my attention to it when he said that the under-correcting of myopia was a greater sin than over-correcting it.

The refractionist employing homatropine should remember the variations found under the several cycloplegics which Dr. Bothman has reported

here. He should be ready to urge his patients to submit to cycloplegia with atropine if he has reason to believe that his findings under homatropine are inaccurate, or if symptoms of accommodative strain persist following the wearing of glasses. Atropine still is the most complete cycloplegic.

Dr. Richard W. Wilkinson, Washington, D. C.: Some twenty years ago I made a series of studies of fifty cases of refractive errors with the use of homatropine and atropine. The conclusion was that there was found a greater refractive error with the atropine in quite a number of the cases, but this defect was usually under half a diopter. The amount of astigmatism and the axis of astigmatism I found varied but little. The conclusion I came to is that all subjects under 16 or 18 should be atropinized, as the difference in the two drugs showed greater in the younger patients. The history of the case may have a great deal to do in determining which drug we should use. In other words, if we have a patient even above 20 who having made the rounds and has not been satisfactorily fitted, or at least is still having trouble, I think that patient should be atropinized.

As to the use of homatropine, I find that if the homatropine is used the night before and then again in the morning, very much as recommended by the doctor, and in addition an occasional drop of a weak solution of cocaine, the cycloplegia will be satisfactory.

With reference to scopolamine and to the toxic effect of it, we have found that young subjects who come into our office with empty stomachs are more susceptible to scopolamine intoxication than those who have come in after being fed. So it is our custom now, particularly in young subjects, to tell them that they had better eat something as we expect to use scopolamine.

Dr. Alfred Cowan, Philadelphia: Dr. Bothman has brought out some points in his very interesting paper about which I should like to make a few remarks. For instance, it seems to me that we cannot choose any particular age for a determination of the kind of cycloplegic to use. It depends on what we want to use the cycloplegic for. If we want a profound and prolonged cycloplegia, there is nothing better than atropine. If we wish to do a refraction with the least inconvenience to our patients with a satisfactory result, we should choose the drug which will give them the least inconvenience. Homatropine seems to answer the purpose of a good cycloplegic in most persons over, say, 12 or 13 years of age. That does not mean it may be used in everybody over 12 or 13 years of age.

All we need to do to determine whether or not a cycloplegic is giving us what we want it to give is to measure the accommodation at the time we do the refraction. In ordinary refractive cases we use cycloplegics to help us to determine the static refraction. It seems to me, as Dr. Gipner brought out, that the method Dr. Bothman used — fifteen minutes after the last instillation of scopolamine — is rather a short time to wait. I always wait a longer time than ten or fifteen minutes after the last instillation of the fifth or the sixth drop of homatropine in each eye the night before; then

on the morning I do the refraction, six or seven drops at fifteen minute intervals, and the refraction not sooner than half an hour after the last instillation. The accommodation is measured before proceeding with the refraction. If the residual accommodation is no more than a diopter or a diopter and a half or even two diopters, I use another cycloplegic. But if good results can be obtained with a cycloplegic which lasts 24 hours, why should one be used that lasts ten days, even in a boy or girl 13 or 14 years of age?

There is another point that I cannot understand and that is that Dr. Bothman found many cases in which the accommodation seemed to have had the ability to overcome astigmatism. I should like to have him explain this a little more in detail.

Then, there is one other thing he mentioned regarding some cases in which the astigmatism was less under more profound cycloplegia. Now that could be accounted for in only one way, and that is that in the dynamic eye the ciliary body pulls and distorts the lens so as to make it astigmatic, while with profound cycloplegia and the relaxation of the ciliary body, this astigmatism disappeared.

DR. LOUIS BOTHMAN, closing: Dr. Gipner stated that I had not given the weaker cycloplegics a chance, that I should have used more and stronger solutions of these drugs. I began my introduction by stating that I was using them in the way we employ these various cycloplegics in our practice. However, in a small series of cases, I continued the homatropine for a period considerably longer than an hour, used more drops than the usual six and then tested the refractions to learn whether there was any difference. In this group of cases, more homatropine gave no further relaxation than that revealed at the end of the first hour. The same is true of a very few cases in which scopolamine was used. Three drops of scopolamine is as much as I care to use in any eye, even though I had only 3 per cent toxic results in this last series.

Dr. Cowan said, I stated that the astigmatism was less under a more profound cycloplegic. That is true. That was more often the case than not; in fact, the astigmatism disappeared under atropine in quite a few cases though it was present with the other drugs. It was unusual to find it the other way. The cases in which I found insufficient cycloplegia under the atropine are the cases I stressed in presenting the paper.

I have here the record of one patient to which I want to call your attention, the left eye of a boy 16 years of age. Under homatropine there was +0.50 sphere with a 0.75 D. cylinder; axis 105. Under scopolamine we found +1.00 sphere, +1.25 cylinder. That is a half diopter more sphere and a half diopter more cylinder. Under atropine there was +1.50 D. sphere with a +1.25 diopter cylinder, a half diopter more sphere than with scopolamine. In other words, a diopter more sphere and a half diopter more cylinder under atropine than homatropine.

Here is another case in which the homatropine refraction showed, in a 30-year-old woman, +2.25 sphere with +1.00 cylinder, whereas with the atropine the error was +2.50 sphere with a +0.50 diopter cylinder.

This is what I found in about 30 per cent of my previous series. The myopic cases had less sphere and more cylinder. In the hyperopic cases, there was more sphere and less cylinder. I think that perhaps explains why a great many men will frequently reduce the strength of the cylinder on a postcycloplegic examination in hyperopes because under homatropine one finds more cylinder than is present when a more complete cycloplegic is used. The fact that these comparisons show from one-half to three-quarters of a diopter difference between homatropine and scopolamine, even higher in individual cases, and the same change between homatropine and atropine in myopia may explain why the myopics are frequently on postcycloplegic examination given weaker glasses than are found with homatropine retinoscopy.

I have noticed that with atropine there is a considerable less number of so-called scissors movements when you do the retinoscopy than one finds in the eyes with less strong cycloplegic. We knew, of course, that in the beginning atropine was a better cycloplegic than the others. I was interested in comparing how it differed.

A great many of us who have refracted Negro eyes under homatropine know how incomplete the cycloplegic is in cases with dark brown irides. In hyperopic patients, with excellent distant vision, frequently with 20/15 vision, will show a half diopter myopia under homatropine. We see these cases occasionally and, when we use atropine, find the eyes emmetropic or slightly hyperopic. I think that is not so much the color of the iris as it is the individual patient or even the race.

I had the opportunity in Burma of examining some of the nurses between 18 and 20 years of age with homatropine. It was like putting water in their eyes. Even atropine was insufficient for complete cycloplegia. In India, also, a dark-skinned race with brown eyes, one drop of homatropine frequently gave almost complete cycloplegia in this same age group of women. This cannot be said for the Negro. The Burmese are a vigorous race and I think that has a lot to do with the comparative action of the drugs.

BENZEDRINE IN CYCLOPLEGIA
II. FURTHER REPORT

S. Judd Beach, M.D., and
W. R. McAdams, M.D.

PORTLAND, MAINE

This is the report of more definite data upon the use of benzedrine with cycloplegics for refraction, certain advantages of which we pointed out in June.[1] This possibility had been suggested to us by a statement in a paper of Myerson's[2] on the pharmacology of benzedrine, which claims that complete but temporary cycloplegia follows use of it with atropine in attenuated solutions. It acts as though atropine inhibits the circular fibres of the ciliary muscle and benzedrine stimulates the radial fibres. Thus an intense dilatation of the muscle results, with corresponding relaxation of accommodation, but shorter than when atropine is used again and again. We do not understand that it is presence of benzedrine that hastens the recovery, but use of less atropine.

If this principle could be applied to the fitting of glasses it might, we thought, remove the most serious objections to cycloplegia for that purpose. Complaints are chiefly against the repeated instillations for cumulative effect, and the consequent protracted recovery. Tried in our clinics, the cycloplegia resulting from a single instillation each of benzedrine and atropine appeared essentially as complete as would be anticipated from cumulative dosage of atropine alone, and of vastly shorter duration. We also found a similar action with homatropine, passing off with astonishing rapidity. Both seemed satisfactory for refraction. Our experiments also indicate a synergism with scopolamine and with euphthalmine.

Results were gauged by retinoscopy with the eye fixing at different distances, and more certainly, by comparing static refraction at distance and near. With intelligent patients, amplitude was measured with a +3 addition at 33 cm. With homatropine, the effect upon one eye of a single instillation followed by benzedrine was also compared with that upon the fellow eye, produced in the orthodox way by multiple administrations without benzedrine.

The present report compares the action of the two methods upon the same eye. Single instillations of the cycloplegics with benzedrine were used first, and the effect measured. When it had worn off, repeated doses without benzedrine were administered in the conventional manner.

TECHNIQUE

We have tried several methods. We began with our regular office solutions. These are 1% atropine and 5% (not 2%) homatropine. The benzedrine (betaphenylisopropylamine or benzylmethylcarbamine) was a 1% aqueous solution of the sulphate.*

In our previous work, one of us had used benzedrine first and the other used it after the cycloplegic. The results seemed identical, but because patients mention tasting the benzedrine almost instantly, we suspected that it dilates the tear passage by adrenalin-like action. As this might facilitate the escape of the cycloplegic, as well as because the action of benzedrine is fast, we both decided to use it last, three or four minutes after the cycloplegic, and have done so in most of the cases in this report. There has never been the slightest toxic effect from the benzedrine.

RESULTS

Cycloplegia from benzedrine-homatropine is rapid. Often it is well along in 20 to 30 minutes, and at peak in 50 to 70 minutes. The fall begins soon so that the examination should be under way within the hour. The recovery progresses rapidly, so that afternoon patients have claimed to read at seven or nine in the evening, and many by eleven. They can usually return to work the next morning. Atropine-benzedrine is also apt to reach its height in an hour, but a small increase may continue for half or three-quarters of an hour longer. Following it, patients have read in one day, but ordinarily require two to four.

MATERIAL

Many patients in our clinics were incapable of the fine discrimination needed for this study. Fortunately Miss Virginia Dudley of the State Bureau of Social Welfare, and Mrs. Katherine M. Dougherty of the Cumberland County Public Health Association became interested in obtaining this unusually thorough examination for their patients. Their following is from intelligent country

* Furnished by courtesy of the manufacturers, Smith, Kline and French Co., Philadelphia.

stock and cooperates well. This enabled us to verify our retinoscopy by trial case tests. We have confined our figures to such patients. There is nothing remarkable about age levels or types of error. The majority of atropine patients were from 8 to 11 years; for homatropine 15 to 19, with scattered cases above and below. The majority needed correction under +4. D., about half of those being under 1 D. Neither these, nor errors of +8. D., nor of considerable myopia, nor variations in age showed any consistent relation to the results.

The figures in these tables are subject to the inexactness affecting all data upon cycloplegia, although obtained under rather favorable conditions. Many variables influence comparisons of the efficiency of different drugs in controlling the ciliary muscle. Age, physical condition, previous use of cycloplegics, and wearing of correction may modify the results, but unfortunately without uniformity. It is difficult to conceive of any satisfactory weighting to equalize such observations. Moreover, wavering of attention is a mischievous source of error in estimation of depth of cycloplegia. Measurement of residual accommodation, both at the trial case and with the retinoscope presupposes concentration of gaze at a fixed distance. This is most difficult to secure in children, for whom these studies are especially designed. Infinite patience is needed for even approximate accuracy.

Yet it may probably be assumed that in the long run the errors more or less even up, so that in general the trend is fairly indicated. There is one exception. In nearly all cases, it was convenient to use the benzedrine method first. This may have given some advantage to the conventional cycloplegia which followed.

Values are computed by rating a cylinder as ½ the worth of a sphere of the same number. Thus a .75 cylinder offsets a .37 sphere. This is analogous to using a cross cylinder and, of course, has the same effect upon both methods.

TABLE I — HOMATROPINE

Depth of cycloplegia following conventional homatropine compared with that induced by the benzedrine method

GAIN		LOSS
+.75 D.....1 case	No change — 7 cases	—.50 D.....2 cases
+.50 D.....1 case		—.25 D.....4 cases
+.37 D.....1 case		—.12 D.....1 case
+.25 D.....3 cases		
+.12 D.....5 cases		

Average gain by use of conventional homatropine 0.035 diopter.

TABLE II — ATROPINE

Depth of cycloplegia following conventional use of atropine compared with that previously induced by the benzedrine method

GAIN		LOSS
+.75 D.....1 case	No change — 4 cases	—.87 D.....1 case
+.62 D.....2 cases		—.25 D.....2 cases
+.50 D.....6 cases		
+.37 D.....2 cases		
+.25 D.....6 cases		
+.12 D.....3 cases		

Average gain by use of conventional atropine 0.23 diopter.

Offhand, the predominance of small plus shifts in the atropine group and the rather non-committal homatropine figures suggest that the cumulative use of atropine is likely to abolish about .25 D. more activity of the ciliary muscle than benzedrine-atropine, but that with homatropine there is little to choose. This, of course, conforms to the common experience, confirmed by Bothman in the preceding paper,[3] that prolonged atropinization gives the utmost relaxation, while homatropine is less reliable. We are not yet ready, however, for a final decision. Moreover, for several reasons, accuracy to .25 D. is not to be expected in a study of cycloplegia. The error due to irregularities in the media uncovered by mydriasis affects the precision of retinoscopy and of subjective tests. Probably most disturbing is the variation in instillation. It is a precarious method of drugging, due to activity of the lids and to tears. With the benzedrine method where everything hangs on one drop, this adds to the difficulty of obtaining uniform administration. Multiple dosage has the advantage that the more drops that are instilled, the greater is the chance that one will be effective.

MODIFICATIONS OF TECHNIQUE

Recently, when we have had reason to believe that the first drop had been flicked, squeezed, or washed out of a hypersensitive eye, we have used a second one following the benzedrine. The slight anesthetic property of the benzedrine seems to make subsequent instillations better tolerated; hence, it may turn out that best results follow use of benzedrine first. This is to be distinguished from the repetition of the cycloplegic or of the entire treatment at the end of an hour, which we used sometimes to employ when accommodation seemed to persist, often followed, in the case of atropine, by a measurable increase in effect. Of course, this would

have been likely to follow without the extra drop, so we abandoned the practice, deeming it unnecessary. Also, because with homatropine, inconsistently, the effect was as apt to diminish, even when we went ahead and used six more drops of homatropine in the conventional way.

We have experimented with several other methods of administration. To avoid the rare reaction in sensitive patients to even a single drop of atropine, we have been trying higher dilutions. Our experience is too small to report, but we find solutions of 1:500, both of atropine and of benzedrine, worthy of further study. In some instances it has seemed possible to intensify the cycloplegia following prolonged atropinization at home, by instilling atropine and benzedrine, in addition, a half an hour before examination. This may turn out to be a powerful method of inducing cycloplegia. Satisfactory cycloplegia also follows the repeated use of benzedrine mixed with homatropine, six doses in an hour, after the analogy of cocaine and homatropine. It is kinder to the cornea and does not require an hour and a half of closed eyes to prevent exfoliation, but the recovery is slow.

It is imperative in using the benzedrine method to make certain of proper instillation of the cycloplegic. Benzedrine itself is so potent that a minute drop in contact with the cornea seems adequate. In using the cycloplegic, however, whether but once, or both before and after the benzedrine, we try to avoid loss by placing the head so as to incline the cornea toward the horizontal, and by controlling the lids to prevent squeezing or snapping it out. Too much should not be expected of a single drop unless properly instilled.

CONCLUSION

The benzedrine method has, with us, had a special field of usefulness in the care of out-of-town patients, and in the clinic. When time is short the action is quick. No potent drugs need be given incompetent parents. Systemic effect causes no anxiety. Cycloplegia is possible where a second visit cannot be made for examination, and where students and adults must return immediately to work. Patients enthusiastically welcome the freedom from annoyance, and the rapid recovery. Where results are uncertain, the orthodox method can still be employed.

To insure effective instillation, we favor use of the cycloplegic, either 5% homatropine or 1% atropine, followed in two or three

minutes by an instillation of 1% benzedrine; then, allowing a couple of minutes for this to take effect, a second instillation of cycloplegic.

At present, it is our feeling that in critical cases prolonged atropinization is probably justified to compel utmost reaction, although it is not as reliable as generally believed. In general, the gain over the benzedrine method is so slight and uncertain that it is doubtful whether it often compensates for the loss of time.

BIBLIOGRAPHY

1. Beach and McAdams: *Benzedrine in Refraction. Preliminary Report.* Tr. A. O. S., 1937 (in press).
2. Myerson and Thau: *Human Autonomic Pharmacology: IX.* Arch. Ophth. v. 18, No. 1, p. 87.
3. Bothman: *Refractive Errors in the Same Eyes While Under the Effects of Homatropine, Scopolamine, and Atropine.* Tr. Am. Acad. of Ophth. and Otolaryng. 1937, p. 165.

DISCUSSION

DR. HUNTER H. MCGUIRE, Winchester, Va.: We should feel deeply grateful to Dr. Beach, whose work in refraction problems has been so outstanding, for calling our attention to a new method of inducing cycloplegia which gives less discomfort to the patient, hastens the period of recovery and, at the same time, seems to yield as accurate results as were obtained by the older methods.

The work of Myerson and Thau, who proved conclusively that benzedrine was synergistic in its action with atropine, prompted Dr. Beach to use this preparation, both with atropine and homatropine, in obtaining paralysis of the accommodation in his refraction cases. The results of his experiments were embodied in a preliminary report which he made at the June meeting of the American Ophthalmological Society and which is, as yet, unpublished. His conclusions, though incomplete at that time, were so convincing that I determined to give the method a thorough trial in private patients during the past summer.

As the 1% solution of benzedrine for ophthalmic use was not marketed at that time, there was an unavoidable delay in obtaining the preparation. My observations have, therefore, been made over a period of only two months and have been limited to about two hundred cases in private practice. It was manifestly impossible, in this class of cases, to take the time to compare the two methods, but Dr. Beach, who has had an opportunity to do this, has shown conclusively, in his work with clinic patients, that

complete cycloplegia is just as readily obtained with a single instillation of benzedrine in combination with either of these drugs as by repeated instillations without benzedrine. Many of the cases which I examined during the past summer had been refracted by me over a period of years by the conventional method and in these I was astonished to find that the accommodation was just as effectually abolished by the new method as by the old.

In order to make a fair comparison of my own results with those of Dr. Beach, I purposely used in all cases a 1% solution benzedrine sulphate with either 5% homatropine hydrobromide or 1% atropine sulphate.

The technique of instillation was likewise according to his suggestions. One drop of benzedrine was first instilled and followed in four minutes by either homatropine or atropine. The patient was then allowed to wait for one hour at the end of which period the refraction was begun. It is most important, in my opinion, that the examination should not be delayed longer than one hour, particularly when homatropine is used, for in several instances in which there was an unavoidable delay of a half hour or longer it was evident that the accommodation was beginning to be reestablished.

The first effect of benzedrine is a rather marked hyperemia of the conjunctiva with, in some cases, though not in all, an intense lacrymation. This usually passes off quickly and when the patient returns for the examination, the eye is invariably white and free from irritation. The refractive results were always confirmed with the retinoscope and the abolition of accommodation determined by the usual methods.

The time element in the reestablishment of the accommodative power was variable but always much shorter than with homatropine or atropine alone. All patients were requested to report either verbally or by letter the exact hour and date when fine print could be read without discomfort. While this information was not obtained in all cases, there were many reports in which accurate data could be recorded. In the cases in which homatropine was used with benzedrine the longest period of recovery was 27 hours and the shortest 4 hours. Most of the patients who had been refracted during the morning hours were able to read in the late evening and a great majority of those examined in the afternoon hours returned to work the following morning. When used in conjunction with atropine there was also considerable variation in the length of time required for a return of the accommodation. The longest period of recovery was 6 days and the shortest 2 days. In most instances recovery was complete in 3 or 4 days. The amount and type of the refractive error and the age factor seem to have little influence upon the length of time necessary for the reestablishment of the accommodation. Eyes with high degrees of error appeared to recover about as quickly as those in which the error was trivial.

In no case was I able to discover any corneal disturbance, rise of tension or systemic effect.

My own conclusions, though based upon a much more limited experience, are in entire accord with those of Dr. Beach. The distinct advantage of being able to induce complete cycloplegia, under supervision in one's own office, in the shortest possible time and the definite shortening of the period of recovery should appeal to both physician and patient. Thus far, my experience convinces me that the method will stand the test of time.

Dr. Conrad Berens, New York: Anything which saves the time of the busy ophthalmologist and adds to the accuracy of his work is welcome; therefore, we and our patients are indeed fortunate in being able to use benzedrine as suggested by Beach and McAdams.

For the past twenty-two years we have used disks containing $\frac{1}{50}$ gr. of cocaine and $\frac{1}{50}$ gr. of homatropine instead of the repeated instillations of homatropine. Therefore, repeated instillations have not been a problem in our practice.

However, our study of benzedrine and homatropine showed that repeated instillations of atropine and homatropine are unnecessary except in a limited number of cases which is a vitally important point for those who use repeated instillations. Our researches also have confirmed the statement that the cycloplegic cycle is of shorter duration with the benzedrine as an adjuvant than from the repeated administration of atropine and homatropine. In determining the residual accommodation, we studied the amplitude of accommodation, after the addition of plus spheres to the manifest refraction, while reading the smallest print (photographically reduced) that the patient could see. We have previously shown (Manual of Med. Res. Lab., Air Service Division of Military Aeronautics, Washington, D. C., 1918) that in studying accommodation the use of print of this character is almost as accurate and more generally applicable than the Duane line.

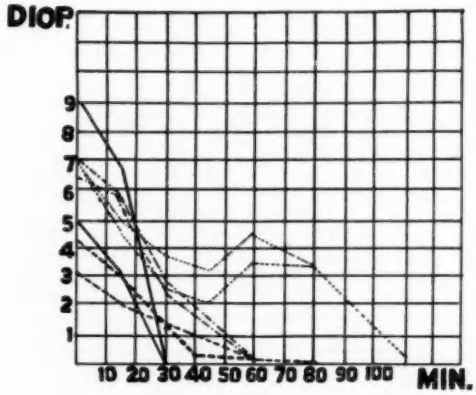

Fig. 1 — (Berens) — Action of benzedrine (1%) and homatropine (5%) usually complete between 30 and 60 seconds.

Cycloplegia from benzedrine-homatropine is rapid. Often accommodation is markedly paralyzed within 20 to 30 minutes, and in the majority of cases is almost completely abolished in from 30 to 60 minutes. The residual accommodation did not exceed 0.25 D. in 70 per cent of the 23 eyes studied (Fig. 1). In this respect the results are similar to those obtained with the orthodox method of using atropine. The use of atropine in 1 per cent solution three times a day for three days caused a reduction of only 0.25 D. in accommodation when compared with one instillation of benzedrine and homatropine. After the use of homatropine and benzedrine most patients may use their eyes for reading within three to six hours.

Doctor Loutfallah (to whom I am indebted for these measurements) and I have had only two failures following the instillation of benzedrine. Both patients required additional instillations of homatropine (5%) after 45 minutes (Fig. 2).

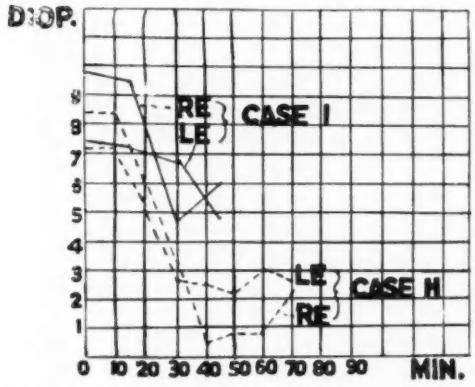

FIG. 2 — (Berens) — Case 1 — Almost complete failure after 45 minutes. Case 2 — Partial failure, L.E. more than R.E.

The minimum effect on accommodation from homatropine and benzedrine was noted in a myopic boy 19 years of age who accepted a —2.25 sph. in each eye. At the end of 45 minutes the right eye retained 6.0 D. of accommodation and the left eye 4.8 D. The second patient was a girl, 18 years of age, who accepted a +.50 D. cyl. axis 90° in both eyes. The residual accommodation was right eye, 2.3 D., and left eye, 2.6 D. It might be advisable to instill benzedrine and homatropine twice unless accommodation can be studied after 45 minutes.

With the use of homatropine and cocaine in disk form the paralysis is as complete and the action in our hands slightly more certain than with the benzedrine and homatropine drop method but the disk method has two serious disadvantages: The need for the use of 0.5 per cent eserine solution (two instillations) if recovery is to be rapid, and clouding of the cornea. The retinoscopic shadow is markedly clearer with the benzedrine method. The need for a cycloplegic which acts rapidly and from which recovery is almost immediate is supplied definitely by benzedrine in combination with homatropine and atropine apparently without deleterious local or general effects.

We believe that the benzedrine method has certain advantages over any other method we have used but that further study is desirable. We wholeheartedly recommend the method of Beach and McAdams to our confreres.

DR. W. B. LANCASTER, Boston: This excellent paper by Dr. Beach is a model of the proper method of determining the value of a new drug. As this is the last paper and the statistics have been somewhat tedious, I may be pardoned if I introduce another feature into the discussion. It seems to

me that these two papers make it fair to raise the question, What is the purpose of this cycloplegia about which so much is said and done? If we took some eyes made of glass and brass and had them examined by different men, we should expect to get results that were equal from the different examiners to two or three places of decimals. Why is it then that if we examine human eyes we differ? The obvious answer is because the eyes have an accommodation variable which changes the refraction, therefore, in order to get consistent answers we must abolish the accommodation.

The question, however, is not so simple as this. There are certain other factors. In statistical papers, it is very important to consider the possibility of sources of error, lest there be deception by the statistics, perhaps even of the man who makes them. Self-deception is not too rare. I may be an example of it.

I should like to show two or three slides to illustrate what I think is a source of error in the argument presented in favor of cycloplegia in all cases, or nearly all cases. One of the speakers said it is wrong to measure the refraction of any young patient without using cycloplegia.

This represents a glass lens, and if we imagined a pupil sending off these peripheral rays, the central ray was gotten into focus here. The question is where the retina should be to get a satisfactory image. Well, can you decide in here to a millimeter just where the best point is?

Here we see a cross-section at this point, point B; here is a cross-section at A, and here is a cross-section at C. The farthest circle is at A, but if instead of a bright point of light we had a contrast such as we get with letters, a black letter on white paper, all this peripheral part would be below the threshold and be invisible, and you would get a better image at this point than you would at this point.

Here is an example of the human eye. You see that these rays come to focus at this point, and these two rays come to a focus here, and these come here, and in the cross-section at right angles to that, they come at a focus here and here, and if you add all the oblique sections you will see that you will have a convergence of rays with many foci, each small area of the cornea giving a different focus from the other. Now what does the eye do about that sort of thing? You see they are measured here, myopia at this point, emmetropia here, hypertropia here, myopia 2.75, myopia 2 diopters, myopia 3½, the difference in the refraction in the different areas of the cornea. What the eye does in those cases with that convergence of confused foci is to pick out the point that gives the best vision in a given case.

Dr. C. W. Rutherford, Indianapolis: Very recently, at my request, Dr. Beach kindly furnished information on the use of benzedrine in refraction. The paper implies the authors' impression that the drug or method which demonstrates the most plus or the least minus correction is the most reliable. Comparisons are drawn after using benzedrine-homatropine at one examination and homatropine alone at another. It would seem desirable to have a scale by which each result could be measured or weighed and conclusions drawn accordingly, as far as conclusions can be based on subjective responses.

A number of years ago, Dr. R. J. Masters of Indianapolis explained to me the method of estimating the degree of cycloplegia and I have used it routinely since. A plus 3.00 D. sphere is added to the distance correction already in the trial frame; letters whose dimensions subtend angles of five minutes at a distance of 50 centimeters are made to slowly approach the patient's eye alongside a Prince's rule until they become legible, and this point is noted on the rule. The far point will be located at 33 centimeters, where the correction is accurate and cycloplegia is absolute. Where the far point is located beyond 33 centimeters it may be inferred that there is an undercorrection for distance. Where the far point is located at less than 33 centimeters it may be inferred that cycloplegia is incomplete and that the patient is accommodating; also it can mean that there is an overcorrection for distance. A far point located between 30 and 37 centimeters suggests that cycloplegia is within about 0.25 D. of absolute.

Since receiving the method of procedure with benzedrine, I have used it in the preparation of twelve patients; their ages ranged from 18 to 51 years. Blepharospasm was partially controlled by flowing over the cornea a drop of anesthetic (one per cent pontocaine) a few minutes before instilling the cycloplegic solutions. Homatropine was used in the conventional manner in the right eyes and homatropine-benzedrine in the left ones. The average far point for the series was 32 centimeters for both right and left eyes, or one-eighth diopter. These figures do not differ materially from those obtained in former cases where homatropine was used alone.

Patients have read news print in from twelve to thirty hours sooner with the left eye than with the right. The method has made a most favorable impression.

If the far point test could be proved to be a reliable guide there would be no practical advantage in making a second examination, as the authors have done. If the test would show that the homatropine-benzedrine method provides adequte preparation, the patient would have the advantage of a shortened recovery period after cycloplegia and there would be less antagonism to the use of drops.

Dr. William Crisp, Denver: I entirely agree with Dr. Lancaster as to the tendency of the eye to select the part of the pupil which by experience it finds most useful and practicable for accurate vision, and I agree with him also that this is a very good reason for not relying too completely upon retinoscopy for our final regimen as to the refraction of the eye.

However, I cannot agree with Dr. Lancaster in one further point which he made, and that is that when the pupil is artificially dilated you necessarily have brought into play the element of spherical aberration at the edge of that pupil. It is my strong impression that the eye continues to select even when the pupil has been artificially dilated, and that, therefore, you are not including in your measurement, as a rule — there may be exceptions — the more hypertropia of the pupil as compared with the center which the eye normally uses.

I do feel that the statement as to the selection of suitable pupillary area should be borne in mind much more generally than it usually is by those

who make a retinoscopic examination, and then check very briefly at the trial case, but essentially rely upon retinoscopy. I think that is a very misleading practice.

DR. WARREN M. DODGE, Battle Creek, Mich.: I have two things to discuss. First, I have used homatropine and benzedrine for five months, since hearing of it in Washington, just before Dr. Beach presented his paper in Atlantic City. I find that it carries out almost continuously his statements. I have been disappointed only two or three times.

The second thing is that I think Dr. Lancaster is right, but if one follows the teachings of my old professor, Dr. Burch, and in doing retinoscopy watches for the simple bright shadow in the center of the pupil and disregards the other areas, retinoscopy will be much more accurate, and you have used Dr. Lancaster's point and you have disregarded the peripheral rays that converge before or behind the point of fixation.

DR. S. JUDD BEACH, closing: There is nothing to add to what has been said except to thank the discussers for the interest which they have added to the paper. Much of their work has been of a quality to make a paper in itself.

I do want to say this work is still in the experimental stage and we have no thesis to urge one way or the other; also that any work done with it should be carefully selected, as has been suggested repeatedly this afternoon, by measurement of the accommodation. This I think ought to apply to all cases in which cycloplegia is done.

I was astounded in making a study a couple of years ago of 100 case reports submitted to the American Board of Ophthalmology in refractions to find that only 13 per cent of the men made any check of the completeness of their cycloplegia. Gentlemen, this shows a pathetic trust in the efficiency of cycloplegia, and sooner or later you are sure to be betrayed.

FILTER PASSING AGENT AS A CAUSE OF ENDOPHTHALMITIS*

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CLARA M. MCKEE
BY INVITATION
BALTIMORE

IT HAS long been known that certain supposedly virus diseases, notably herpes zoster, may occasionally produce intraocular inflammatory complications, but the possibility of a virus causing endophthalmitis as an isolated symptom in man has not, heretofore, been indicated. Current opinion is inclined to regard all cases of uveitis as produced either by some specific infection, such as syphilis or tuberculosis, or as resulting from a focus of bacterial infection elsewhere in the body. While the present report represents the results of a study of an isolated and, perhaps, in some respects, peculiar case of uveitis, the indication that uveitis, even as an isolated symptom, may be caused by a filter passing agent is not without interest. It seems desirable, therefore, to report in some detail the results of both clinical and laboratory studies made on even this single case.

CASE REPORT

In the autumn of 1935, a young white woman, aged 25, was admitted to the Wilmer Institute, complaining of ocular pain and loss of vision. Her family history was entirely negative. Her parents and six siblings were all living and well. Her general health had always been excellent. She had had the usual childhood diseases without complications. She had never had any serious injuries nor any operations. She had few upper respiratory infections; no headache nor toothache, nor any trouble with her ears. There was no history of cardio-respiratory, gastro-intestinal, genito-urinary, or neuro-muscular disease. Her habits were regular and she took no drugs.

* From the Wilmer Ophthalmological Institute of the Johns Hopkins Medical School and Hospital, and the Department of Immunology of the Johns Hopkins School of Hygiene and Public Health.

Six weeks prior to her admission her eyes had become red and inflamed and during the next week she began having photophobia and lacrimation, but the symptoms were not severe until two weeks before admission when she noticed severe aching in her eyes and headaches, generally worse at about 4 a.m., and associated with considerable blurring of her vision. For a short time prior to her admission the vision in the left eye had improved slightly.

On admission, her temperature was 99.6° (rectal), pulse 100. General examination aside from the eyes was entirely negative.

There was very slight swelling of the lids and moderate conjunctival and pericorneal congestion with marked tenderness to pressure over the ciliary region in each eye. The pupils were already partially dilated by a mydriatic but were reported previously to have reacted normally. Examination with the corneal microscope revealed, in each eye, that the cornea was normal, but that there were large numbers of cellular deposits on its posterior surface. The anterior chamber was of normal depth but the aqueous ray was heavy. The iris appeared œdematous, somewhat congested, with the normal markings partially obliterated. There was one small posterior synechia in each eye. Vision was: Right 20/70, Left 20/40, not improved by lenses or pinhole. Visual fields showed normal peripheral outlines, but a marked enlargement of the blind spot in each eye. Ophthalmoscopic examination revealed a fine vitreous dust slightly blurring the fundus details. The discs were quite congested with somewhat blurred margins, physiological cups were obliterated but there was little elevation of the discs. The retina surrounding the discs appeared œdematous, the vessels in some places being obscured. The arteries were narrow, veins full and tortuous, no hemorrhages or exudates were seen.

In the effort to discover the cause of this condition a great number of examinations were performed. Examination of the nose and throat revealed an hypertrophic left tonsil but no evidence of focal infection. There were no dead teeth, nor evidence of oral sepsis. Neurological examination revealed no involvement of any nerve other than the optic. Examination by gynecologist disclosed no genito-urinary disease.

Laboratory tests revealed normal blood chemistry, including sugar, non-protein nitrogen, uric acid, calcium, cholesterol and phosphorous. There were 4,900,000 R.B.C.; 5,700 W.B.C.; hemo-

globin was 74%; differential count showed P.M.N. 79%, juvenile neutrophiles 2%, lymphocytes 15%, large mononuclears 4%, no eosinophiles or basophiles. Urine examination was negative save for occasional W.B.C. in the sediment. Intra-cutaneous tuberculin test was positive to 1 mg. O.T. but negative to lower concentrations. Intra-cutaneous uveal pigment test was negative, blood Wassermann negative, blood culture negative, agglutination test for typhoid and mellitensis negative. X-rays of the sinuses, teeth and chest revealed no evidence of disease.

Two weeks after admission, lumbar puncture was performed. There were 70 cells, all mononuclears, per cubic millimeter in the spinal fluid. A second lumbar puncture one week later showed 40 cells. At this time the spinal fluid was cultured, some was injected into a guinea pig, and an anti-formin concentration was done. All these procedures gave negative results. A third tap one week later again revealed 70 cells and a fourth tap one month after the third revealed 22 cells. Spinal fluid Pandy, Wassermann, and gold curve were negative.

The patient showed little change during her first two weeks' stay in the hospital. She had a slight, somewhat irregular fever with afternoon peaks that reached as high as 101° F. on one occasion. There was slight relative tachycardia. At the end of two weeks, the temperature and pulse rate both began to return to normal. At about the same time the external ocular congestion decreased. The aqueous ray became less intense and the posterior synechiæ began to break up. A week later the papilloedema showed some diminution but on the fourth week after admission, the patient had a mild relapse with increased ocular congestion, fresh synechiæ and increased blurring of the optic disc. It was not until two months after admission, three and a half months after the onset, that a decided and permanent improvement set in. The patient was discharged two and a half months after admission, her vision having returned to 20/20 in the right eye, 20/30 in the left. Two months later her vision was 20/15 in each eye. The fundi showed faint areas of choroidal depigmentation about the discs, and there had been no relapse in her condition. The patient was last seen in the summer of 1936, at which time no further change was noted.

The spinal fluid obtained from the third and fourth taps was used for animal inoculation.

EXPERIMENTAL

The spinal fluid obtained in the third lumbar puncture was bacteriologically sterile. Within one hour after its removal it was injected into three rabbits as follows: Rabbit 1 received 0.1 cc. of the fluid in the vitreous of the right eye, 0.2 cc. intracranially, and 0.2 cc. intratesticularly. Rabbit 2 received the same intraocular and intracranial injections. Rabbit 3 received the same intracranial injection and in addition scarification and inoculation of one cornea. There was no reaction to the intracranial, corneal, or testicular inoculations. The animals all remained in good general health and showed no neurological symptoms. Rabbit 2 showed no reaction to the intraocular injection beyond that attributable to trauma. There was minimal conjunctival congestion on the day following the inoculation but this cleared in 24 hours and no further developments were noted, though the animal was kept under observation for two months. Rabbit 1 showed no reaction for four days following inoculation. On the fifth day the inoculated eye became slightly congested. There was a faintly positive aqueous ray and one small synechia. The vitreous was slightly hazy and there were extensive ill-defined greyish-white areas in the periphery of the retina associated with small retinal hemorrhages. On the following day the vitreous haze had increased slightly. Fluid was aspirated from the vitreous and inoculated into another rabbit. This fluid was bacteriologically sterile. Further animal passages were made in series as described below.

The spinal fluid obtained from the fourth lumbar puncture of the patient was bacteriologically sterile on culture; 0.1 cc. of the fluid was inoculated into the vitreous of one eye of each of three rabbits. One of these rabbits showed no reaction to the inoculation but the other two developed lesions similar to those seen in rabbit 1. From each of these one or more passages were made, yielding positive results.

ANIMAL PASSAGES

Rabbits

The infection obtained in rabbit 1 was transmitted in series through seven successive sets of rabbits. During the first three of these passages the method of transfer was as follows: A small amount of fluid was aspirated from the vitreous of the infected eye and injected directly without filtration into the vitreous of another rabbit. The aspirated eye was then removed and ground with

broken glass. A small amount of salt solution was added, and the emulsion was filtered through a Berkefeld V filter, and the filtrate inoculated into the eyes of one or more animals. It was found that the fluid aspirated from the vitreous of infected eyes within the first six days after inoculation was capable of producing the infection on inoculation into another animal, but the material aspirated later in the course of the disease was no longer infectious. On the other hand, the ground up ocular tissues after filtration through a Berkefeld filter were found to be infectious even in the third or fourth week of the disease, and this method alone was used in further passages.

In all, fourteen rabbits were inoculated with such filtrates and all showed substantially the same clinical course as that described in regard to rabbit 1. On the first day after inoculation, there was usually an insignificant traumatic reaction which disappeared by the next day. After a short symptomless interval, fresh lesions were seen which usually made their appearance on the fourth to seventh day after inoculation. At this time the infected eyes usually showed slight pericorneal congestion and a positive aqueous ray. About one-half of the infected eyes showed synechiæ. There was at this time some haze in the vitreous, often diffuse, sometimes dusty, sometimes in the form of fluffy grey masses. At the same time the optic disc was congested and its margins hazy. Retinal lesions which were present in every successful transfer were usually found in the lower periphery (opposite the site of injection) and were of two types. Some were small fluffy greyish spots about ½ to 1 p. d. in diameter; others were large, densely white, with soft outlines, often associated with petechial hemorrhages. In many instances both types of retinal lesions were seen.

After the first appearance of these lesions they generally increased in intensity for two or three days and then slowly regressed with occasional relapses and fresh extension of the lesions as late as four weeks after inoculation. In regressing, all of the symptoms tended to disappear but the large white patches in the retina remained longest, and in some instances were still present as late as six months after inoculations.

No constitutional symptoms were noted in the course of the disease. In a few instances the rectal temperature was recorded but found to be normal. In only one instance did lesions appear in the uninoculated eye. This occurred in the one instance in the series in which the Berkefeld filtrate used was found to have become acci-

dentally contaminated with bacteria. While we have not been able to repeat the findings in this experiment, they are of sufficient interest to warrant detailed report.

Rabbit 4, which had been inoculated directly from rabbit 1, showed the usual course of symptoms described above. On the seventh day after inoculation, this animal was killed and fluid aspirated from the vitreous of the affected eye and injected into rabbit 5. The eye of rabbit 4 was then removed, emulsified and filtered, and the filtrate inoculated into the right eye of each of two rabbits, 6 and 7. Subsequently the filtrate was found to be contaminated with a large Gram positive bacillus. Rabbit 5 showed no reaction to the inoculation. Rabbits 6 and 7 both developed purulent panophthalmitis in the inoculated eye in which Gram positive bacilli could be demonstrated on smear and culture. Rabbit 6 showed no lesions in the uninoculated eye, but on the fifth day after inoculation, rabbit 7 showed slight pericorneal congestion in the uninoculated eye, slightly turbid aqueous, several fine synechiæ, diffuse vitreous haze, congestion and blurring of the disc and some large white patches in the lower periphery of the retina. Since in our experience, bilateral reactions to unilateral purulent infections in rabbits have never occurred, we feel that the metastatic lesion in this case was probably due to the filter passing agent. The eye was removed for histological study (Fig. 1). Further successful passages were made from the bacteria-free filtrate of the right eye of rabbit 7.

In view of the relatively mild course of the disease in rabbits, the effort was made to find some other more susceptible animals. At various stages in the course of the rabbit passages, attempts were made to infect rats, mice, guinea pigs, dogs, and cats.

Rats and Guinea Pigs

The Berkefeld filtrate of an infected rabbit's eye was inoculated into each of three rats and three guinea pigs. At the same time some of the fluid was injected into the eye of another rabbit. This rabbit developed typical lesions. A small amount of the fluid was injected intraocularly in one eye of each of the rats and guinea pigs, 0.1 cc. was injected into one testicle of each animal, and 0.1 cc. was injected into the cerebro-spinal fluid just below the foramen magnum. None of the animals developed testicular lesions or constitutional symptoms. Two of the three rats developed an intraocular hemorrhage, probably traumatic, but showed no other recognizable ocular lesions. Two of the three guinea pigs showed on

the fourth day after inoculation slight pericorneal congestion, contracted pupils, and slight haziness of the vitreous. The congestion and pupillary contraction disappeared in two days but the vitreous haze increased slightly for one week and then began to subside. It was concluded that these species were relatively resistant to the infection.

Mice

On three occasions groups of three to ten mice were inoculated intracranially with Berkefeld filtrates of infected rabbit or cat eyes. No symptoms developed attributable to the inoculation. It was concluded that mice are resistant to the infection.

Dogs

After five rabbit passages an effort was made to transmit the infection to dogs. The eye of an infected rabbit was emulsified and filtered through a Berkefeld filter and the filtrate was injected intraocularly into one eye each of one rabbit and two dogs. The rabbit developed typical lesions. In the dogs there was an insignificant traumatic reaction which disappeared on the second day after inoculation. On the next day, however, an inflammatory reaction developed in the eyes of both animals. In one there was moderate pericorneal congestion, positive aqueous ray, vitreous haze, congested optic disc and retina. The inflammatory reaction increased in severity for a few days and then began to subside. By the end of two weeks after inoculation the eye had returned to normal. The second dog showed a much more severe reaction. On the third day after inoculation there was marked pericorneal congestion, numerous cellular deposits on the back of the cornea, hemorrhage in the anterior chamber, slight posterior synechiæ. The vitreous was hazy, optic disc blurred and congested. A week after inoculation the exudate and hemorrhage in the anterior chamber had been partially absorbed and numerous areas of greyish haze could be seen in the retina. Three days later, fluid was aspirated from the vitreous of this eye and inoculated into the eyes of two more dogs. The aspirated fluid was sterile on culture. The reaction to this first transfer in dogs was more severe than that to the initial material derived from a rabbit. In one animal the bulbar congestion was intense. A large hemorrhage appeared in the anterior chamber on the third day and a deep corneal haze subsequently developed. In the other animal, moderate pericorneal congestion, positive aqueous ray, hazy vitreous, congested retina and optic disc, grey spots

in the retina characterized the course of the reaction which persisted for three weeks before beginning to subside. The eye showing the most severe reaction was emulsified and filtered through a Berkefeld filter. The filtrate was inoculated into one eye each of a rabbit, a cat and a dog. The rabbit showed a typical reaction. The dog developed a reaction similar to that of the animal from which the inoculum was derived. The cat developed even more severe lesions.

Cats

The reaction of cats to the infection was so severe that the clinical appearance at times resembled a pyogenic infection. The following technique for passage was, therefore, regularly used. The injected eye was removed, emulsified in saline with broken glass and the emulsion filtered through a Berkefeld or Seitz filter. At the same time the eye of a control cat was removed and similarly treated. The "virus" filtrate and "control" filtrate were each injected into one or more cats for passage. Cultures were made of all filtrates and were uniformly found to be bacteriologically sterile. In spite of all precautions, occasional bacterial infections developed in both the "virus" and "control" series. Filtration, however, always successfully removed the contaminant and on passage the "control" animal showed no symptoms while the "virus" animal showed the characteristic "virus" reaction. In all, 28 passages in series have been made involving a total of 63 cats infected with the "virus" and 28 "control" cats.

The reaction of the cats to intraocular injection was as follows. There was usually a slight traumatic reaction which subsided on the second day. On the 3rd to 7th day after inoculation slight congestion of the eye appeared along with dusty opacities in the vitreous. During the first week the animals often appeared sick, lost weight, and showed fever of 1 to 3 degrees. The inflammation progressed in severity and by the end of the second week extensive synechia and heavy cellular deposits on the back of the cornea were usually present. At this stage, massive hemorrhages in the anterior chamber were often seen. If the eyes were not enucleated, secondary glaucoma commonly developed.

During one period, Seitz filtration was used exclusively for passage and it was found that the severity of the reaction gradually decreased from passage to passage. When Berkefeld filtration was reintroduced, the severity of the reaction reappeared after the second passage and has been maintained since.

Repeated attempts have been made to induce an intraocular reaction without intraocular injection, infectious material being inoculated intravenously, intracranially, or into one eye only. In no instance has there been any clinical evidence of infection of an uninoculated eye, but microscopic sections have commonly shown a very slight polymorphonuclear infiltration in the ciliary body and anterior vitreous in such cases. The "virus" has, however, repeatedly been recovered from the spinal fluid of infected animals and, on one occasion, from the nasal discharge.

Cat 10 showed a typical ocular reaction. Five weeks after inoculation the animal was sacrificed. Seitz filtrates were made of the eye and brain of this animal and each inoculated separately into one eye of cat 12. Both eyes of cat 12 showed typical reactions. Four weeks after inoculation cat 12 was sacrificed. The Seitz filtrate from one eye of this cat was inoculated into one eye of cat 13 while the other eye of cat 13 received an injection of spinal fluid from cat 12. Both eyes of cat 13 showed typical reactions. After 4 weeks, spinal fluid was obtained from cat 13 and inoculated intraocularly into cat 15. This cat showed a typical ocular reaction and in addition a marked nasal discharge. Nasal washings obtained from cat 15 were filtered through a Seitz filter and inoculated intraocularly into cat 17, yielding a typical reaction. The spinal fluid of cat 17 was injected intracranially into cat 18, yielding a mild illness with fever and coryza, but no intraocular reaction. Spinal fluid from cat 18 was injected intraocularly into cat 24, yielding a typical reaction, and further eye to eye passages were made from cat 24.

ATTEMPT AT CULTIVATION OF THE "VIRUS" ON CHICK EMBRYOS

The bacteriologically sterile Berkefeld filtrate of a "virus" infected cat's eye was inoculated onto the chorio-allantoic membrane of an eight-day chick embryo. On the fourth day after inoculation a transfer was made to another eight-day chick and successive transfers were made every fourth day from chick to chick for eight passages. No lesions were seen on the membranes of the first two chick embryos of this series, but after the third transfer small raised plaques appeared on the chorio-allantoic membrane, and this reaction persisted up to the eighth transfer. From the eighth chick embryo bacteriologically sterile material was obtained which was injected into the eye of cat 66. At the same time, material from a normal chick embryo was injected into the eye of cat 67. Both

cats 66 and 67 showed some reaction attributable to the foreign protein but the reaction of cat 66 was more severe than that of 67. The Berkefeld filtrate of the eye of cat 66 yielded severe reaction on transfer and further passages in series were successfully performed, whereas the Berkefeld filtrate from the eye of the control cat 67 produced no reaction on the next or succeeding transfers.

PRESERVATION OF THE "VIRUS"

After several successive passages of the virus by Berkefeld filtration from eye to eye in cats, an effort was made to test the viability of the "virus" in storage. An infected cat's eye was removed 3 weeks after inoculation and the intraocular tissues were emulsified with ground glass in a small amount of saline solution. The resulting emulsion was centrifuged at low speed for five minutes. The supernatant fluid was then poured off and filtered through a Berkefeld V filter. Three cc. of a 50% solution of glycerine in saline was added to the sediment and this was stored under vaseline seal in a refrigerator. A small portion of the Berkefeld filtrate of the supernatant fluid was injected into a cat's eye and produced a strong reaction. The remainder of the Berkefeld filtrate was covered with sterile melted vaseline and stored in a refrigerator.

Three months later, these stored materials were used for inoculation. The glycerinated sediment was ground up in a mortar, centrifuged at low speed for five minutes and the supernatant fluid was injected into the eyes of cat 80. Since the violent reaction which ensued might be attributable to the irritant effect of the glycerine, a further passage was made by Berkefeld filtration two weeks later from cat 80 to cat 84, yielding a typical and severe reaction. Similarly, the stored Berkefeld filtrate, after being kept in the refrigerator for 3 months was injected into cat 81. A mild reaction resulted but further passage produced again the typical severe reaction.

PATHOLOGICAL EXAMINATION

In the course of this investigation, the eyes of many infected and control animals were removed at various intervals after inoculation and subjected to histological examination. Up to the present it has been impossible to demonstrate specific inclusion bodies in any of the material studied. The pathological changes found in the eyes of rabbits, dogs and cats were all more or less similar in form and distribution but showed marked difference in intensity. The least intense reaction was that of rabbits.

In these animals the inflammation of the uveal tract was insignificant. There were serum and a few mononuclear cells in the anterior chamber, and scattered mononuclears in the iris and ciliary body. No lesions were seen in the choroid. The vitreous contained some serum and cellular infiltrations were present near the retina, in front of the optic disc and in the zonular fibres. Scattered patches of round cell infiltration were found in the retina with microscopic evidence of tissue destruction. In some animals, extensive and intense regions of pre-retinal infiltration were seen, corresponding to the large white areas noted ophthalmoscopically. In the cases examined late in the disease these pre-retinal infiltrates were found to have become organized by glial proliferation.

The changes most regularly found in rabbits' eyes and seemingly most characteristic of the infection were in the optic disc. This was swollen both laterally and forward. An accumulation of cells was usually present in the cup and there were perivascular infiltrates in the optic disc and, at times, also in the optic nerve. The one instance of what we regard as a metastatic lesion occurring in the uninoculated eye of a rabbit whose other eye was infected with the "virus" showed all of these features. The perivascular infiltrates in the optic disc were particularly intense and extensive.

In dogs the inflammatory reaction was much more intense than in rabbits. The iris and ciliary body showed extensive diffuse mononuclear infiltrates with occasional nodular accumulations of round cells, either perivascular or in the stroma of the tissue. Fine fibrinous adhesions were found between the iris and lens. In several instances there was a massive hemorrhage in the anterior chamber and a diffuse cellular infiltration in the cornea. There was, however, remarkable lack of evidence of any extensive tissue destruction in the iris and ciliary body, and the choroid was not affected. The vitreous was filled with serum, and many mononuclear cells of all types were present, especially in the pre-retinal portions. Extensive destructive lesions were found in the retina with focal round cell infiltrations. The reaction at the optic disc was intense with massive infiltrates filling the cup, intense œdema and occasional hemorrhages in the dics and surrounding retina, and massive perivascular infiltrates.

In cats the lesions of the retina and optic disc were similar to those in dogs, but, in general, the involvement of the iris and ciliary body was more intense. Large nodular accumulations of lymphocytes were commonly found in these tissues. In the ciliary body

not only the stroma but also the epithelium was involved in the inflammatory reaction, small nodular regions of epithelial destruction and mononuclear infiltration were frequently seen. Those cats with severe reactions that were allowed to survive the illness eventually developed secondary glaucoma with peripheral anterior synechia. The retinal tissue in these cases was completely disorganized and replaced by massive sheets of glial proliferation.

The microscopic examination of the brains, testes, and visceral organs of cats and rabbits did not reveal any lesions attributable to the "virus."

DISCUSSION

The experimental results reported above indicate clearly that a filter passing agent such as is commonly termed a virus has been secured which is capable of causing a severe endophthalmitis in cats and dogs. Owing to the fact that numerous passages were made in rabbits before the infection was transferred to dogs and then to cats, the question may be raised as to whether the disease which we have investigated most extensively in cats is really related to that of the patient from whom the infectious material was originally obtained. It is conceivable that during the passage through rabbits and dogs, some virus, native to these animals, was accidentally obtained, and that it is this virus, rather than one originating from the patient with which we have infected cats. Unfortunately for the purposes of this investigation, the patient had made a complete recovery from her disease before the relatively high susceptibility of cats to the infection had been discovered, and direct inoculation from the patient into cats was, therefore, not done. We have, however, been able to travel part of the road back to the source by inoculating material which had previously sustained many passages through cats back into rabbits and have produced again in rabbits mild lesions clinically identical with those originally produced by injecting the patient's spinal fluid into animals of this species. We believe, therefore, that it is most probable that the virus whose lesions in cats we have studied is actually identical with that obtained from the patient, and that, therefore, the disease from which the patient suffered was due to this same agent. The remote possibility that the disturbance in the patient's eyes and spinal fluid may have been unrelated requires mention.

None of the animals which we have infected in the course of these studies has developed symptoms which we could recognize as characteristic of any known and previously studied virus. The

possible relation of this virus to two others requires, however, further comment.

In 1929, Woods and Chesney demonstrated that "periodic ophthalmia" of horses was produced by a virus. These authors inoculated rabbits intraocularly with the Berkefeld filtrate of the eye of a horse suffering with this disease. Passage was made by Berkefeld filtration from rabbit to rabbit in series, and finally back to a horse, yielding in the horse characteristic symptoms of periodic ophthalmia. The lesions produced by the virus in rabbits were extremely mild, consisting essentially of a slight diffuse haze of the vitreous and plaques of white exudation in the retina. However, inoculation into horses of material obtained from normal control rabbits failed to produce any disease in horses. It is apparent, therefore, that the lesions produced in rabbits by the virus of periodic ophthalmia were clinically similar to those which we have reported here. We have studied the reactions of rabbits to three different strains of periodic ophthalmia virus and have noted a close similarity of these reactions to those produced by the virus which we have been studying. In general, however, the periodic ophthalmia virus causes a much less severe reaction in rabbits than does the virus which we have been studying. We have attempted to demonstrate cross immunity between these various strains but have failed as yet to demonstrate any immunity in rabbits even to strains with which they have been previously infected. It is evidently necessary to pursue these comparisons in other species, but, for the present, we are of the opinion that the two viruses are not identical, though they may be related.

During the past few years a new clinical entity has been described under the name of a benign lymphocytic meningitis and has been shown to be due to a virus. The causative agent of this disease, however, is fatal to mice. The immunity of mice to the virus which we have obtained serves to dissipate any notion that the two might be identical.

SUMMARY

A case is reported of bilateral uveitis with papillitis and increased cell count in the spinal fluid. On animal inoculation of the spinal fluid of this case a filter passing agent such as is commonly called a virus was secured which produced characteristic intraocular inflammatory lesions when inoculated into the eyes of rabbits, dogs and cats. The virus passes the Berkefeld V filter more

readily than the Seitz filter. It can apparently be cultivated on the chorio-allantoic membrane of chick embryos and can be preserved for several months in anaërobically sealed Berkefeld filtrates of infected tissues and in glycerinated emulsions of infected tissues. The virus produces in rabbits lesions somewhat similar to those produced by the virus of periodic ophthalmia of horses, but the evidence at hand would indicate that the two viruses were not identical.

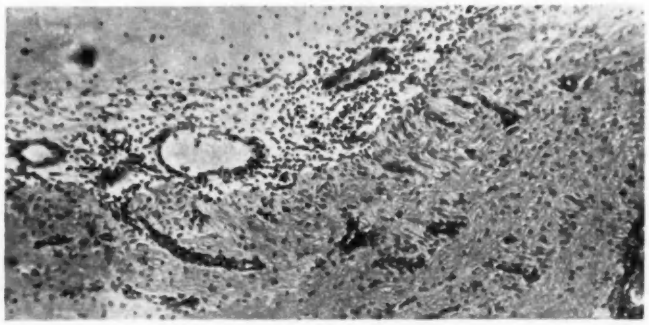

FIG. 1 — (Friedenwald and McKee) — Optic Disc of a Rabbit Showing a Reaction in the Uninoculated Eye.

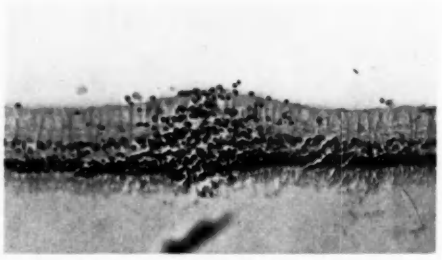

FIG. 2 — (Friedenwald and McKee) — Retina of a Rabbit Showing a Reaction in the Uninoculated Eye.

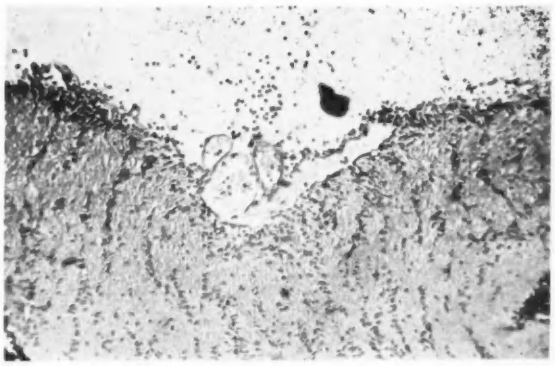

FIG. 3 — (Friedenwald and McKee) — Optic Disc of the Inoculated Eye of a Rabbit.

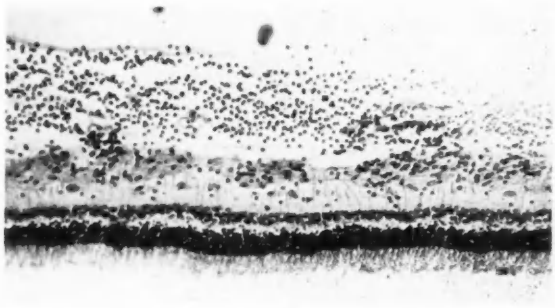

FIG. 4 — (Friedenwald and McKee) — Retinal Lesion Corresponding to a "Snowbank" Exudate in an Inoculated Rabbit's Eye.

FIG. 5 — (Friedenwald and McKee) — Nodular Lesion in the Pars Plana of the Ciliary Body of a Rabbit.

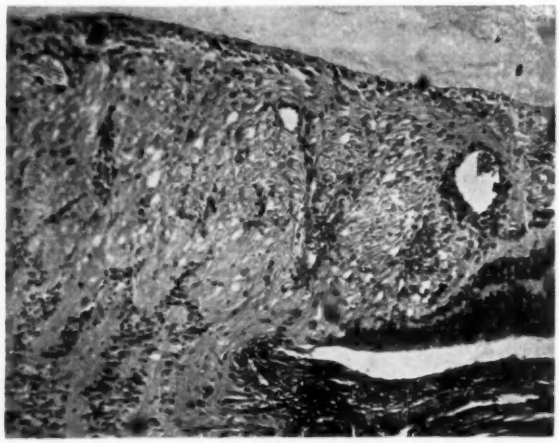

FIG. 6 — (Friedenwald and McKee) — Optic Disc of the Inoculated Eye of a Dog.

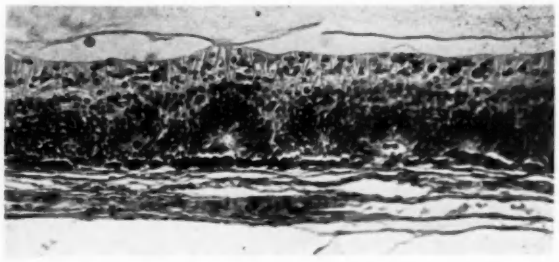

FIG. 7 — (Friedenwald and McKee) — Retinal Lesions in a Dog.

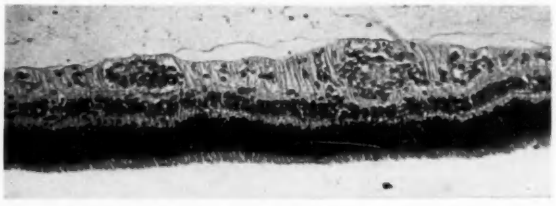

FIG. 8 — (Friedenwald and McKee) — Retinal Lesions in a Dog.

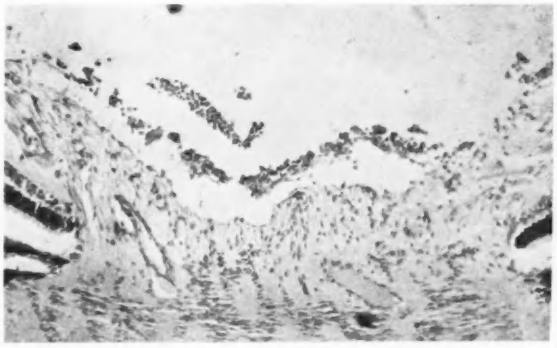

FIG. 9 — (Friedenwald and McKee) — Optic Disc of Inoculated Cat's Eye.

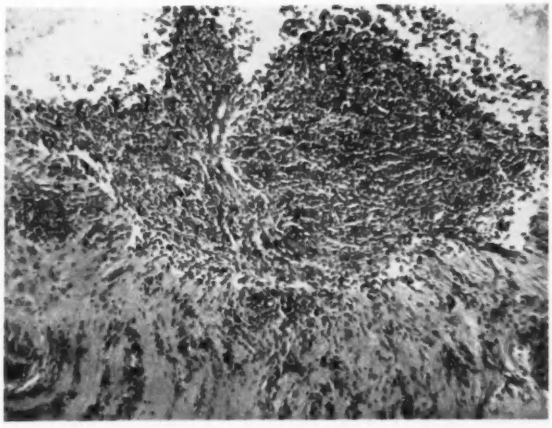

FIG. 10 — (Friedenwald and McKee) — Optic Disc of Inoculated Cat's Eye.

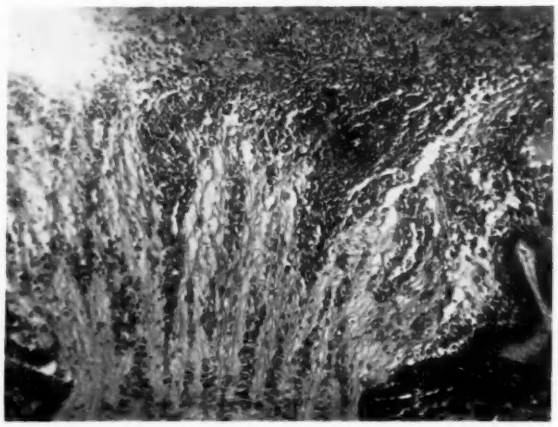

FIG. 11 — (Friedenwald and McKee) — Optic Disc of Inoculated Cat's Eye.

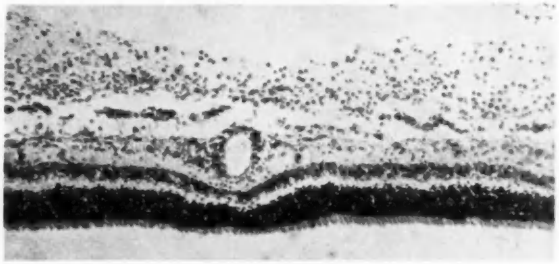

FIG. 12 — (Friedenwald and McKee) — Retinal Lesions in a Cat.

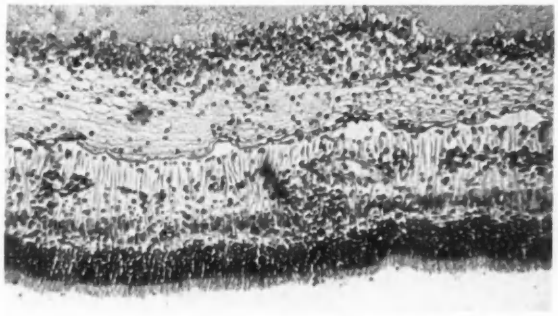

FIG. 13 — (Friedenwald and McKee) — Retinal Lesions in a Cat.

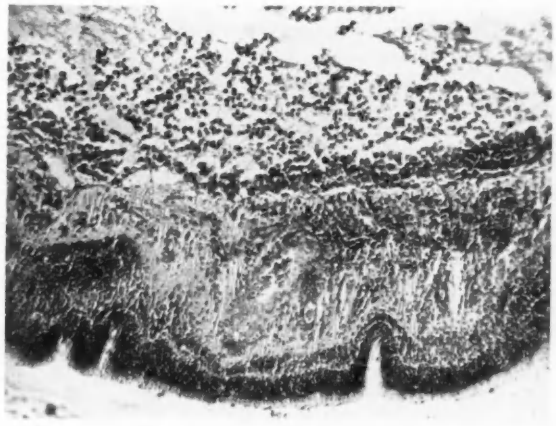

FIG. 14 — (Friedenwald and McKee) — Retinal Lesions in a Cat.

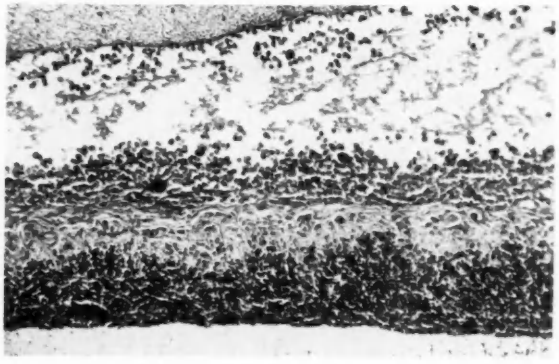

FIG. 15 — (Friedenwald and McKee) — Retinal Lesions in a Cat.

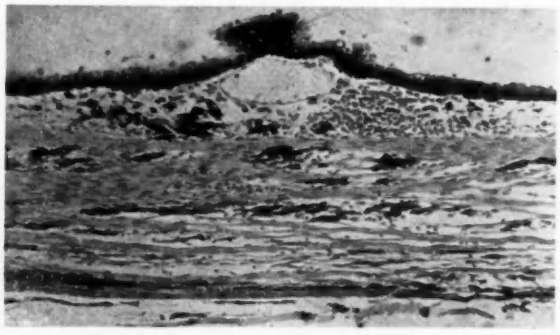

FIG. 16 — (Friedenwald and McKee) — Nodular Lesion in Pars Plana of the Ciliary Body of a Cat.

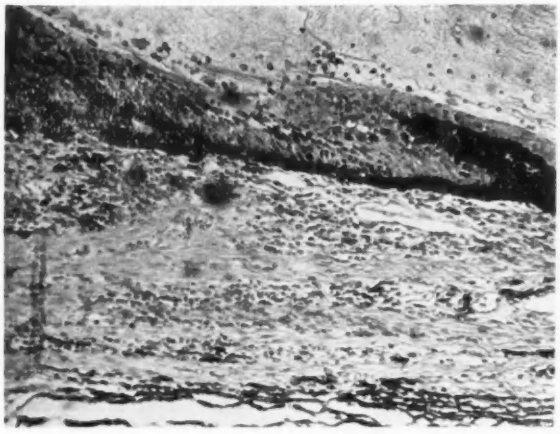

FIG. 17 — (Friedenwald and McKee) — Lesions in the Ciliary Body of a Cat.

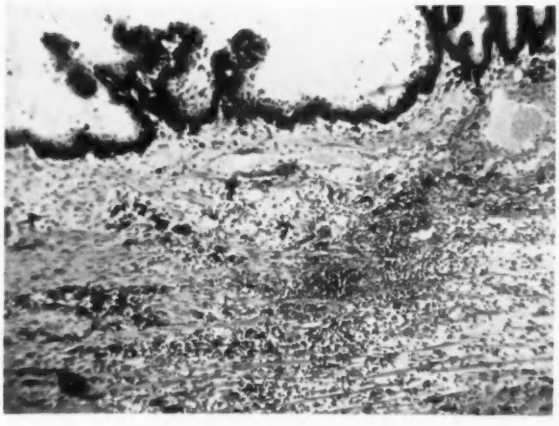

FIG. 18 — (Friedenwald and McKee) — Lesions in the Ciliary Body of a Cat.

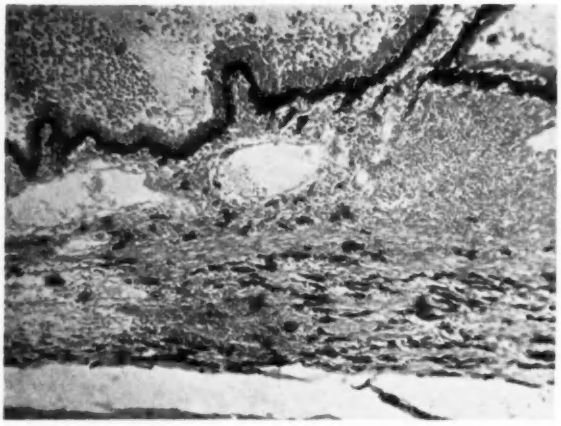

FIG. 19 — (Friedenwald and McKee) — Lesions in the Ciliary Body of a Cat.

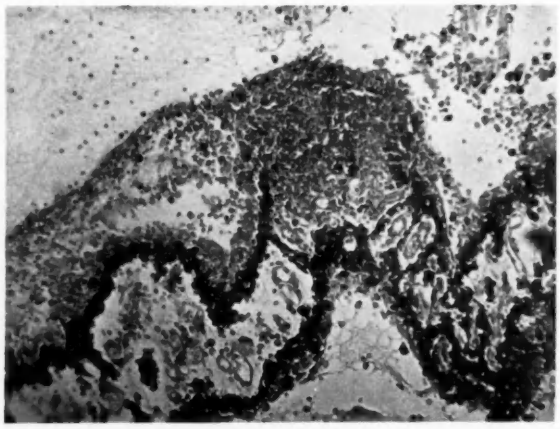

FIG. 20 — (Friedenwald and McKee) — Lesions in the Ciliary Process of a Cat.

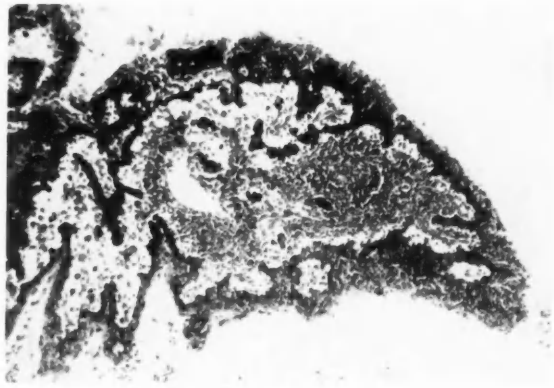

FIG. 21 — (Friedenwald and McKee) — Lesions in the Ciliary Process of a Cat.

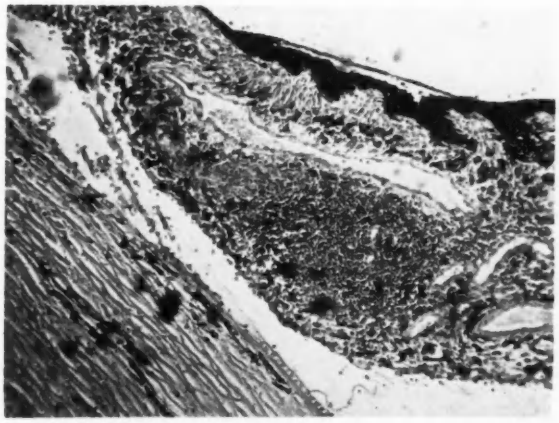

FIG. 22 — (Friedenwald and McKee) — Lesions in the Iris of a Cat.

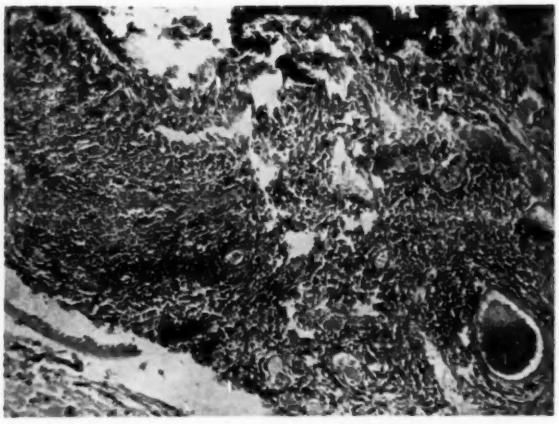

FIG. 23 — (Friedenwald and McKee) — Lesions in the Iris of a Cat.

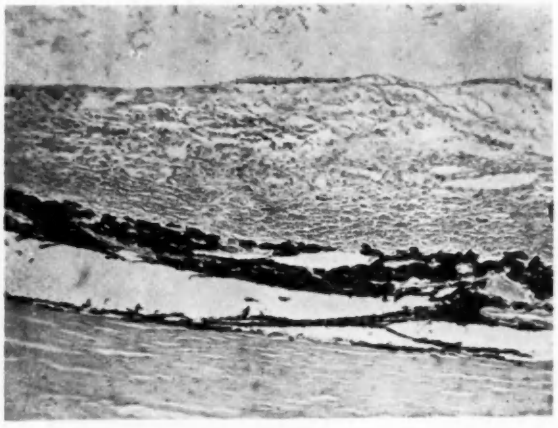

FIG. 24 — (Friedenwald and McKee) — Retina of a Cat's Eye Six Months After Inoculation.

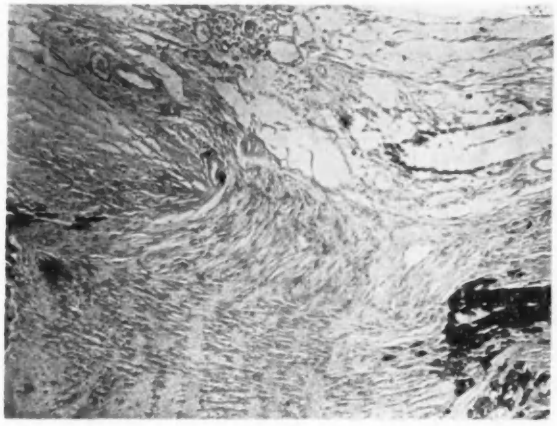

FIG. 25 — (Friedenwald and McKee) — Optic Disc of a Cat's Eye Six Months After Inoculation.

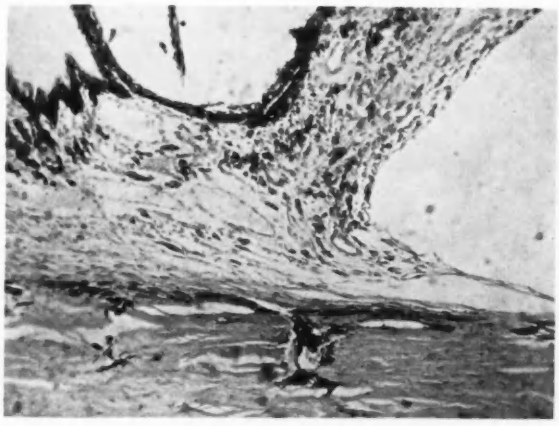

FIG. 26 — (Friedenwald and McKee) — Chamber Angle of a Cat's Eye Six Months After Inoculation.

DISCUSSION

DR. ALBERT L. BROWN, Cincinnati: Dr. Friedenwald's observations concerning the isolation of a filtrable virus capable of producing endophthalmitis upon intraocular injection in animals is of great interest. The report lends itself to discussion in three parts: (1) The virus as an isolated phenomenon, (2) its action on the eye and other organs, (3) its relation to the pathogenesis of human uveitis.

(1) The human case reported is apparently one of uveitis, albeit with certain unusual characteristics. The description of the eyes suggests a composite of bilateral optic neuritis and generalized uveitis, which the essayist wisely calls endophthalmitis. In the absence of all other findings in the patient, a virus was isolated from the spinal fluid which survived many intraocular passages in animals. This may simply attest the vitreous as a favorable medium for the virus, but not necessarily specific properties to produce ocular lesions. (2) The ocular reactions in the various animals were all produced by direct intraocular injection. These reactions, especially in cats, seemed severe endophthalmitic effects rather than any specific tissue entity seen in humans. The lesions, especially those revealed by the microscope, were lodged variously in the retina, optic nerve, and uveal tract. The predominant tissue effects depended upon the species of animals used. Experimentally, the virus could not be said to produce any characteristic ocular pathologic entity. The ocular reactions caused by the virus were well studied but observation of its action on other organs seemed inconclusive. The negative effects of intratesticular and intracranial injections are given in several instances. The results of intrathoracic and intra-articular injections in a fair series of the same species of animals, if negative, would help to affirm an ocular specificity. As a matter of interest, I should like to have known the results of intravenous injection of the virus in animals previously receiving intraocular injection after the reaction had subsided. (3) Repeated extraocular injections of the virus in animals, even in the sensitive cats, failed to produce an ocular reaction. This may have been due to the inability of the virus to survive bodily passage in these animals. The virus may be more hardy in the human. Tests for skin sensitivity of the patient to the virus might have been interesting. The author carefully avoids the assumption that the virus produces uveitis in humans, but contents himself with noting its intraocular effects. This stand seems fair. The evidence of the patient's eyes and the animal injection suggests that the virus is an isolated phenomenon and probably the cause of this individual case of endophthalmitis. Thus, another interesting and puzzling observation has been added to the enigma in ophthalmology, intraocular inflammation.

DR. S. HANFORD McKEE, Montreal: It is unnecessary in an audience of this kind to any more than recall to your minds how frequently, in certain cases of uveitis, routine examinations disregard tuberculosis and other systemic disease, and focal infection is found to be negative, leaving the ques-

tion of etiology entirely obscure and the consequent symptomatic treatment equally unsatisfactory.

Dr. Friedenwald's report will serve to bring to your attention the increasing importance of viruses as a cause of disease in man. The predilection of disease for nervous tissue is well recognized as exemplified in infantile paralysis.

Dr. Friedenwald's report opens a new field as regards the etiology of the disease of the uveal tract and may be the means of throwing much light upon the cause of disease of this area.

The technique of transmission to susceptible animals has been carefully and thoroughly carried out, so that any laboratory with modern equipment should be able to verify the history of this case. It is hoped others will follow the lead Dr. Friedenwald has given us, so that the splendid piece of work may be corroborated.

PERSONAL EXPERIENCES WITH INTRACAPSULAR CATARACT EXTRACTIONS

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THE intracapsular method of cataract extraction is increasing in popularity both in this country and abroad, tho it has by no means supplanted the extracapsular procedure. While some still reject the operation because of convictions concerning the wisdom of removing the posterior capsular diaphragm and the anatomic disturbance of the vitreous body, many have hesitated because of fear of the operation. From the author's limited experience, the latter seems entirely unjustified. The impression has prevailed that the intracapsular method is too hazardous for any but surgeons of very large experience, and many men have avoided the operation for this reason. No doubt prejudice against the intracapsular extraction has been due to early unfortunate results reported in this country following the original method introduced by Colonel Smith. With the more refined and gentle technic of forceps extraction this should be dispelled.

This paper reports the results of 200 consecutive intracapsular cataract extractions performed by the author and associate staff of the Wisconsin General Hospital during a period of four years. About two-thirds of the cases were operated upon by the author personally, the remainder by associates, assistants, and resident staff members. Thus, in considerable number, the first intracapsular operations of the resident staff members are included. This point is stressed in order that the reader may properly evaluate the results.

The operations herein reported were performed by the combined forceps and expression technic, similar to the methods originally introduced by Stanculeanu, Knapp, and Torok, and modified by Lancaster, Safar, Elschnig, and others. There is, however, a definite variation or modification of their procedures, to be de-

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scribed later. It is beyond the scope of this paper to enter upon a detailed description of the evolution of the forceps expression method of intracapsuler extraction. This has been adequately covered by Lancaster.[8]

There was no rigid rule for the selection of cases for operation, tho complicated ones, such as high myopia, those in which there was an increase in intraocular tension, fluid vitreous, or posterior synechiæ were usually tho not invariably reserved for extracapsular extraction.

PREPARATION FOR OPERATION

Complete physical examination, with Wassermann tests, were made on practically all patients. All foci of chronic sepsis were removed where possible. This pertained especially to abscessed teeth, pyorrhea, etc. In those cases where the lachrymal sac showed secretion or there was a history of recurrent infection, extirpation was performed prior to operation.

Smears from the conjunctiva of the lids were invariably made, and operation was rarely performed unless they were negative for bacteria and pus cells.

Some 18% of the patients were operated under avertin anæsthesia, the technic of which has been discussed by the author elsewhere.[1] Those patients operated under local anæsthesia received gr. 1½ nembutal by mouth one-half hour prior to operation. Euphthalmine, 3%, one drop was instilled into the conjunctival sac one hour before operation. The eye-brow was shaved or clipped.

OPERATION

Preparation on the operating table consists of the usual thoro cleansing of the skin about both eyes, and painting a 3% tincture of iodine on the skin of the lids, brow, and nose. This was usually wiped off with 70% alcohol. The conjunctival sac is thoroly irrigated with boric acid solution, and the lids cleansed with 1:5000 bichloride solution. Cocaine, 4%, five to six drops is instilled into the cul-de-sac over a twenty-five minute period. One or two drops of adrenalin, 1:1000, is instilled just before operation. Van Lint akinesia, with 2% novocaine, is generally used, the orbicularis at the internal canthus also being injected. One drop of 1% cocaine was injected subconjunctivally at the limbus below. Retro-orbital injection of novocaine, injected thru the skin along the floor of the orbit, was used in about twenty-five patients, but this is reserved for special cases.

A speculum was invariably used thruout the operation, Skeel's modification of Weeks' being preferred in most cases.

Fixation of the globe was made at the lower margin of the internal rectus muscle at its insertion, using ordinary fixation forceps with a catch. A bridle suture under the superior rectus muscle was used in about half of the cases. A Graefe incision of 35% to 45% of the corneal circumference was used with the addition of a conjunctival flap. The size of the incision was adapted to the type of lens to be delivered. The section followed the limbus as closely as possible with a moderate size conjunctival flap. Two silk sutures were inserted immediately after the section and drawn aside. Usually two or three more sutures, sometimes four, were added after the end of the operation. Sutures are of the straight thru and thru type of very fine braided silk (Deknatel N004 B 1 test) tied in three knots. Carrel arterial needles, size B 21, full-curved, were used for the conjunctival suturing.

A small iridectomy was made in most cases. A buttonhole peripheral iridectomy was made in the simple extractions before delivery of the lens.

EXTRACTION OF THE LENS

The capsule of the lens was grasped at the junction of the lower and middle thirds. Elschnig's and Arruga's forceps were used in most of the first 100 cases, and the author's forceps in the remainder.

After grasping the capsule, the forceps are held as steady as possible with no rocking movement whatever. Counter-pressure is then applied at the limbus below with the edge of the ordinary cataract spoon. The pressure is directed backward toward the optic nerve, and is gentle and in no way comparable to the force formerly exerted in the Smith operation. Slight motions sidewise, with the spoon, to either side of 6 o'clock, are made directly backward until the zonule ruptures and the lower edge of the lens appears to luxate forward, after which the pressure is upward and a little backward, the forceps being synchronously withdrawn as the lens advances upward thru the wound.* The forceps are never deliberately pulled upon, except as pressure is applied below and they are then withdrawn as the lens advances upward thru the in-

* This method of delivery of the lens was first demonstrated to me by Dr. William L. Benedict, to whom I acknowledge my indebtedness. I have been unable to find any published description of the exact technic employed, tho it corresponds closely to that described by Lancaster in his discussion before this Society in 1933.

cision. The lens tumbles in most instances, tho frequently it may be delivered head on, depending upon the nature and size of the nucleus. The forceps aids in bringing the lens thru the wound and also in breaking the upper zonular fibers. Gradle[2] speaks of his method of intracapsular delivery as 80% push and 20% pull. This procedure might be described as 95% push and 5% pull. After delivery, the sutures are immediately tied and additional sutures inserted. Following this, the iris pillars are replaced.

Should the capsule rupture coming thru the wound and should there be a question of some remaining, the capsule forceps are again introduced just inside the lips of the wound and any remaining capsule withdrawn. Should the capsule rupture before the lens is delivered, an ordinary extracapsular procedure is carried out, first making sure that a large portion of the capsule has been removed.

Pilocarpine, 2%, was instilled into the eye in all cases at the close of the operation, eserine 1% being added in the simple extractions. Bichloride of mercury ointment, 1:5000, was placed between the lids and especially over the lachrymal puncta. Vaseline gauze patches were placed over the lids, and both eyes were lightly bandaged for four days.

POST-OPERATIVE TREATMENT

The first dressing is done on the fourth day in cases in which complete iridectomy has been performed and after forty-eight hours in cases of simple extraction. Patients are kept in bed four to five days, a back-rest being permitted after forty-eight hours. A soapsuds enema is given on the fourth day. Sutures are removed on the seventh day unless spontaneously extruded. The unoperated eye is uncovered on the fourth day after operation, and the operated eye is dressed on alternate days until dismissal which, in private cases, is usually the tenth or twelfth day. In ward cases the average stay in the hospital is fourteen days. Atropine, 1%, is used at the post-operative dressings, if needed, but frequently only one or two instillations are required since reaction is usually so slight after the intracapsular procedure.

Ward cases were refracted at the end of two weeks for a visual record, but glasses were not prescribed until six weeks later. The records show a number who failed to return to the Hospital for final refraction, so the vision recorded is that on discharge. Our records show the vision was usually improved at the end of six weeks.

STATISTICAL ANALYSIS OF TWO HUNDRED INTRACAPSULAR EXTRACTIONS

The age of the patients is shown in Table 1, being grouped in decades from 40 to 90. (The youngest patient operated was 42 and the eldest 87.)

TABLE 1

AGE	
40 to 50 years	10 cases
50 to 60 years	34 cases
60 to 70 years	65 cases
70 to 80 years	82 cases
80 to 90 years	9 cases
	200 cases
SEX	
Males	109 cases
Females	91 cases
	200 cases

The state of maturity of the cataracts in this series is shown in Table 2: 125 were classed as mature, 58 as immature, and 17 as hypermature. I have found it profitable to carefully study each lens after removal by tearing off the capsule and examining the cortex and nucleus. In this way we are increasing our ability to judge more exactly the nature of the cataract before operation and thereby choose the proper procedure and estimate the size of the section for removal in a given case.

TABLE 2—*Description of Cataracts*

Mature	125	Sclerosed	58
		Ordinary senile	67
Immature	58	Amber nucleus, clear cortex	21
		Ordinary immature, including post-subcapsular	37
Hypermature	17	Shrunken	15
		Fluid cortex	1
		Rubbery capsule	1
	200		200

With regard to the primary cause of the cataracts, I am unable to offer any important evidence. Nineteen patients had diabetes, in varying states, tho only six of these showed diabetic retinitis and only two were considered diabetic cataracts. Two cases had high myopia. These cases are usually operated by the extracapsular procedure, but due to the marked immaturity of the cataracts in these two instances the intracapsular method was employed. Five patients had chronic simple glaucoma, in one of which the eye had been trephined ten years earlier, with complete control of the tension. This case, incidentally, was extracted with complete success, with the capsule intact and with no loss of vitreous. There was one allergic cataract. In one case a detachment of the retina had existed probably eight years. A preoperative presumptive diagnosis of detachment or intraocular tumor was made, and an extensive detachment was observed on the operating table immediately after removal of the lens, discernible by oblique illumination. Several cases showed subluxation of hypermature cataracts, with vitreous in the anterior chamber, and these will be referred to later. In one case there had been an old, mild, plastic iritis, with slight posterior synechia.

TABLE 3 — *Method of Operation*

Preliminary iridectomy	103 cases
Combined extraction	87 cases
Simple extraction with peripheral iridectomy	10 cases
	200 cases

Iridectomy, either preliminary or at the time of extraction, was performed in most cases, as shown in Table 3. Personally, unless there is some very definite indication to the contrary, I prefer the combined extraction, but others on the service prefer preliminary iridectomy. There is no occasion to discuss the relative merits of this practice here, as these are too well known, and various surgeons differ in their preference. The simple extraction gives the ideal post-operative result when successfully performed, but from the standpoint of safety and simplicity of extraction, complete iridectomy has a definite advantage. The ten cases of simple extraction were usually younger subjects and were all highly successful from an operative and visual standpoint, tho prolapse under the conjunctival flap requiring excision occurred in one case.

TABLE 4 — *Condition of Capsule on Delivery*

Capsule intact	164 cases (+ cases)	82%
Lens + Capsule (capsule broke thru wound but was completely removed)....	36 cases (+ — cases)	18%

The cases in which intracapsular extraction was attempted but failed are not included in this group, as these fall in the class of extracapsular extractions. Records have been accurately kept, however, of the course of these cases. We were successful in extracting 79% of our cases in capsule in which the operation was attempted.

Rupture of the capsule in my hands has been most common in the completely sclerosed lens, which is large, hard, and brown, with no subcapsular cortical material. The capsule appears thin and friable and does not move freely on the underlying cortex, as seen in many lenses, since none is present. Tumbling is difficult due to the fact the lens will not mold. Fortunately these cases are ideal for extracapsular delivery, due to the absence of soft cortex, tho I usually attempt to remove them in capsule. In the glistening, slightly intumescent lens, especially where the capsule is under slight tension, it ruptures easily when grasped with the forceps.

The hypermature lens dislocates most easily, tho, when of the morgagnian variety and swollen and distended, with rubbery capsule, it is practically impossible to grasp it even with Fuchs's toothed forceps. I have removed some of these quite easily in capsule by external pressure only.

The immature lens, with clear cortex and amber nuclear haze, is ideal for extraction. The capsule moves freely on the underlying cortex, which one may demonstrate by rubbing the extracted lens between the thumb and forefinger. The capsule, therefore, is easily grasped without rupturing due to this and to the presence of ample pultaceous subcapsular material. The loose capsule can be seen projecting up thru the jaws of the forceps. This feature of the forceps which I have designed has been discussed before the American Ophthalmological Society,[3] where they were last year exhibited. The large area of capsule which can be grasped distributes the traction over a good portion of the equator of the lens.

The fully mature, homogeneous, grayish-white lens is usually easily delivered, the capsule holding well.

As a general rule the operation is most successful in the age group from 60 to 90 years, since the zonule appears weakened and easily breaks. One is surprised at times, however, by the ease of extraction of the soft, white, rapidly developing cataract which is seen in the age group from 40 to 50 years, if time is allowed for a full, deep chamber.

PROLAPSE OF VITREOUS

A record of cases in which vitreous was lost, with the last recorded vision, is shown in Table 5.

TABLE 5 — *Vitreous Loss — Analysis of Cases*

Vitreous appeared in the lips of the wound or was lost in varying amounts in twelve cases or in 6% of the total number operated.

	Vision	*Capsule*
1. Small amount after delivery	20/15	intact
2. Fluid, small amount flowed out during operation	20/20	intact
3. Weak zonule; very little loss; lens fell back on grasping; lifted out in capsule	20/15	intact
4. Tiniest amount; shrunken lens (15 years' duration) lifted out with forceps in capsule	20/200	intact
5. Very small amount after delivery; poor co-operation	20/20	intact
6. Moderate amount; lens fell into vitreous; delivered with loop	20/30	intact
7. Tiniest bead; appeared after suturing wound on replacing pillar	20/30+	+ —
8. Moderate loss after delivery	20/30	intact
9. Four to five drops; after delivery	20/20	intact
10. Tiny drop; after delivery	20/30	intact
11. One drop; after delivery	20/40	intact
12. Two drops; after delivery	20/30	intact

As noted, there was a loss of vitreous in varying degrees in twelve cases, or 6% of the 200 operations. This is less than that in another series of 100 extracapsular extractions which were operated upon by substantially the same group (aside from changing house-officers) during the same period. In the latter series there was a 9% vitreous loss. While this is contrary to the usual experience, Gradle[2] reported similar results in 200 cases, 100 intracap-

sular and 100 extracapsular, all of which he had personally operated. His records show 3% vitreous loss in the intracapsular and 7% loss in the extracapsular series, tho there were but 80 intracapsular deliveries.

In the cases recorded in Table 5, it will be noted that vitreous loss was followed by poor vision in but two cases; namely, No. 4 and No. 11. In case No. 4 the amount of vitreous loss was insignificant, and the lowered acuity of vision could not be attributed to this tho the exact cause of such was difficult to determine since the retina was essentially normal. It was an old, hypermature, shrunken cataract of 15 years' duration, in which the iris was tremulous and the vitreous degenerated, tho not fluid. The lens fell back slightly when the capsule forceps were applied. Delivery was easily accomplished by re-introduction of the forceps, and after grasping the capsule again the lens was lifted out intact. No loop was used nor was pressure exerted on the globe with the spoon. This method of delivery of a subluxated lens has not been sufficiently stressed. It is far superior to the older method of loop and spoon delivery in which a considerable amount of vitreous may be lost.

In Case 11 a drop of vitreous was lost. The healing was prompt, but the visual record on discharge on the fourteenth day was only 20/40. The patient has never returned for final refraction.

If the zonule does not break readily with moderate pressure on the globe, we usually abandon the effort and remove the capsule with the Fuchs's capsule forceps.

With the possible exception of inexperience in some of the house-officers, vitreous seems to appear without apparent cause and when least expected. My own experience has been similar in extracapsular cases. Vitreous may appear when there has been a minimum amount of trauma, occasionally after the section, and at times upon replacing a pillar, or when the patient suddenly moves the eye. It would seem that it must be related to the condition of the hyaloid and the consistency or condition of the vitreous itself. In other words, some cases seem predestined to this complication, while every surgeon is familiar with types of cases in which excessive manipulation and unusual trauma are not followed by prolapse. Unquestionably the thoro paralysis of the orbicularis by novocaine injection has reduced this hazard by the complete elimination of squeezing, and large losses seem to have vanished. In my earlier cases we used avertin when we suspected the patient

might be a "squeezer" or a "bad actor," but with increasing confidence in the operation and a more thoro akinesia we have found its use less important.

It is, of course, unnecessary to stress the importance of measuring intraocular tension with the tonometer in every cataract case prior to operation. The intracapsular procedure is usually avoided if tension cannot be brot well within normal limits.

IRIS PROLAPSE

This complication has occurred in a very limited number of this series, as is shown in Table 6.

TABLE 6 — *Iris Incarceration and Prolapse*

1.	Prolapse one pillar 11th day (over-activity at home)
2.	Prolapse after simple extraction (post-operative vomiting)
3.	Prolapse one pillar (patient struck eye)
4.	Prolapse one pillar (anterior chamber hemorrhage; wound sprung)
5.	Caught pillar; no prolapse
6.	Caught pillar; no prolapse
7.	Caught pillar; no prolapse
8.	Caught pillar; no prolapse

As noted above (Case 1) prolapse of one pillar occurred on the eleventh day. The patient had had an exceptionally quiet eye and was dismissed on the tenth day. Too great activity bending over caused one pillar to prolapse thru the wound, without loss of the anterior chamber. It was excised with prompt recovery.

Case 2, a simple extraction, showed a prolapse, completely covered by the conjunctival flap, at the first dressing. The patient vomited several times during the first night of operation and during the next day, from no apparent cause as the operation was under local anesthesia. Excision was followed by prompt recovery with little reaction and excellent vision.

Case 3 bumped her eye, with prolapse of one pillar.

Case 4 had an anterior chamber hemorrhage, in which the wound was sprung, with incarceration of one pillar beneath the flap, requiring excision.

Cases 5, 6, 7, and 8 showed one pillar caught in the wound but required no interference since the eyes were quiet.

HEMORRHAGE INTO ANTERIOR CHAMBER

Hemorrhage into anterior chamber was observed at various dates during post-operative period from the fourth to the fourteenth day. In some of the cases its occurrence was definitely associated with trauma, such as turning in bed with pressure on the eye, getting out of bed and falling to the floor, and on straining from an enema, but there were a number which seemed to occur spontaneously without injury of any kind. Most of them were small and cleared up promptly, but several were of considerable amount, completely filling the anterior chamber and in two causing a bulging of the flap with external oozing. The relative frequency of this mishap is puzzling aside from those directly due to trauma. Col. Wright,[4] in his masterly series of lectures on cataract appearing in the American Journal of Ophthalmology, states in his third lecture that he has met with this accident in over 10% of his cases and, in some years, it has been as high as 20%. No better description of the varied aspects of this complication can be found anywhere. Detailed analysis of our cases will be found in Table 7.

TABLE 7 — *Post-operative Hemorrhage into Anterior Chamber*

No.	*Day*	*Description of Hemorrhage*	*Cause of Hemorrhage*	*Final Vision*
1.	9th	Into anterior chamber	Unknown	20/15
2.	5th	Into anterior chamber	Unknown	20/15
3.	6th	Into anterior chamber	Unknown	20/20
4.	6th	Very slight into A.C.	Unknown	20/20
5.	6th	Into anterior chamber	Rolled onto eye	20/15
6.	6th	Into anterior chamber	Tore off bandage, coughing uncontrollable (same for other eye, extracapsular)	20/40
7.	6th	Into anterior chamber	Unknown	20/15
8.	5th	Severe, vitreous and A.C.	Bumped eye, prolapsed iris excised	Hand Movements
9.	4th	Small into A.C.	Unknown	20/20
10.	4th	Small into A.C.	Fluid vitreous; no loss	20/15
11.	17th	Small into A.C.	Struck eye	20/20
12.	6th	Into vitreous and A.C.; quiet vitreous hemorrhage	Unknown (similar hyphæma in other eye 6 mos. later)	20/30
13.	8th	Into anterior chamber	Vitreous loss at op.; blood remained 5 weeks	20/30
14.	?	Quiet vitreous hemorrhage	Complicated cataract; fluid vitreous	20/30

The above table reveals twelve post-operative hemorrhages into the anterior chamber, one of which invaded the vitreous, and two quiet hemorrhages into the vitreous, for a total of 7% of the cases operated. These results are surprisingly similar to the average of 7.6% reported by Vail[5] in statistics gathered from various sources and presented in his thesis, "Hyphæma after Cataract Extraction." Our experience also confirms this writer's conclusion that the source of the bleeding in most cases seems most likely from the wound rather than the iris.

In my experience, post-operative hemorrhage occurred quite as frequently in cases operated by the extracapsular method, so it does not appear to be due to the intracapsular procedure. Hemorrhage has not been definitely associated with diabetes in this series of cases, tho I have observed a number of such cases previously operated by the extracapsular method which seemed to be related to a diabetic condition.

Our study reveals no definite cause for the bleeding aside from post-operative trauma. A poor section, too scleral, appears definitely to predispose to post-operative hemorrhage. None occurred in the simple extractions. We observed two cases of quiet hemorrhage into the vitreous, discovered only at the end of two weeks when refraction and ophthalmoscopic examination was undertaken. These cleared up satisfactorily, tho vitreous streamers remained. Only one case, No. 8, resulted in marked reduction of vision and this was a post-operative hemorrhage into the anterior chamber and vitreous on the fifth day due to trauma.

VISUAL RESULTS

Table 8 shows the visual record. It should be noted that most of the patients operated upon come from distant parts of the state, so that in many instances the vision recorded is that taken following a brief refraction on the day of discharge, usually the fourteenth day. Some of these cases have their final refraction by local physicians in their home towns, tho an effort is made to have all return for a thoro refraction and glasses at the end of six weeks to two months.

TABLE 8 — *Visual Record*

Vision	*No. Cases*	*Percentage*
20/15	82	83% (20/30 or better)
20/20	54	
20/30	30	
20/40	8	17% (20/40 or less)
20/50	7	
20/70	5	
20/100	5	
20/200	6	
Hand movements	2	
Unknown	1	

As shown above, 83% of the cases obtained 20/30 or better vision, while 17% are grouped 20/40 or poorer. In our extracapsular series of 100 cases, 20/30 or better vision was obtained in 71% of the cases.

In Table 9 will be found an analysis of the intracapsular cases in which 20/40 or poorer vision was recorded. Eight of the 34 cases have failed to return for final refraction and our visual records are those of the day of discharge, usually the fourteenth.

Lowered acuity from vitreous loss could not have been a contributing factor in but two cases in this group, and in one of these the visual record of 20/40 was that on date of discharge — the fourteenth day. One case with vision of 20/200 had an old, hypermature, shrunken lens of fifteen years' duration, with vitreous in the anterior chamber. The retina appeared normal, and vitreous loss was slight. Vision, however, could not be improved.

Aside from these cases and one with hemorrhage into the anterior chamber and vitreous, lowered acuity was chiefly due to preexisting disease, such as high myopia, chronic glaucoma, diabetic retinitis, retinitis pigmentosa, and macular degeneration. In one patient, both eyes were operated with perfect post-operative results, but only 20/200 vision was obtained. The eyes appeared normal. The patient spoke no English and was of extremely low mentality.

TABLE 9 — *Analysis of Cases with Vision of Less than 20/30*

Vision 20/40	
Did not return for refraction (vision on discharge)	3
Chronic glaucoma, preoperative	1
Myopia	1
Hyphæma (tore off bandages)	1
Hypermature lens	1
Cause unknown	1
Vision 20/50	
Diabetic retinitis	3
Chronic glaucoma, subluxated lens	1
Macular degeneration	1
Did not return for refraction (vision 14th day)	2
Vision 20/70	
Retinal hemorrhages — hypertensive retinitis	1
Hypermature lens (15 years) — squint	1
Macular degeneration	1
Did not return for refraction (vision 14th day)	2
Vision 20/100	
Corneal dystrophy	1
Hypermature lens (15 years' duration)	1
Diabetic retinitis	1
High myopia (20D) — squint — vitreous change	1
Did not return for refraction (vision 14th day)	1
Vision 20/200	
Vitreous in A.C. — slight loss — complicated cataract (15 years' duration)	1
Diabetic retinitis	2
Retinal detachment (8 years pre-operative; observed on table)	1
Unknown (perfect intracapsulars; normal fundi)	2
Vision less than 20/200	
Retinitis pigmentosa	1
Hemorrhages, vitreous and anterior chamber, 5th day post-operative	1
Vision unknown	
Death, pneumonia	1
	34

A few other features deserve comment.

One patient, aged 82, developed scarlet fever with otitis media on the sixth day post-operative with no interruption or complication in the healing process, and obtained 20/20 vision. One other

patient had a mild otitis media. Six cases were disorientated and two might be classed as post-operative mania. All cleared up promptly by removal of the patch from the unoperated eye and by getting them out of bed. Several cases had delayed restoration of the anterior chamber, some as late as the ninth day. Watchful waiting, rest in bed, and the use of pilocarpine, 2%, daily was the only treatment employed, and all finally healed without further complication. Late bulging of the wound under the flap occurred in a few cases but there was never actual prolapse of vitreous. External oozing occurred in two of the cases with hyphæma. True iritis was observed in but three cases and none of these were severe. In view of the statements of Knapp[6] concerning this complication, I have searched our records for cases which might come in this category but can find only three which were so classified. The fact that fourteen days was the average length of hospitalization corroborates this in a way, since all patients were kept in the hospital until their eyes were quiet. Some cases show hospitalization of five weeks but for the most part these patients had hemorrhages. No doubt some degree of iris irritation accompanied some of these, but frank iritis was exceptional.

Post-operative vomiting occurred very infrequently — occasionally when no post-operative sedative whatever was used. We scrupulously avoid the post-operative use of morphine, tho it is used with scopolamine, gr. $\frac{1}{200}$, as a preliminary sedative in those cases operated under avertin anæsthesia. Codeine, gr. $\frac{1}{2}$, and aspirin, gr. v, is the only post-operative sedative used, and this is avoided when possible.

Slitlamp study is carried out on all cases returning for refraction after six weeks. The anterior surface of the vitreous occasionally shows a bulging forward thru the pupillary area, and rarely a rupture of the hyaloid is seen. At times holes are seen in the hyaloid, and pigment dust from the iris or blood pigment is occasionally observed. As a rule it appears smooth and flat. Vitreous opacities and fine streamers were seen in a number of cases, tho we have made no statistical study of these. So many cases show this condition before operation, an accurate record would be difficult to obtain.

The number of patients in whom hypertensive cardiovascular disease was recorded was very large. This condition rarely appeared to interfere with a satisfactory post-operative result.

Astigmatic records conform so closely to the many observations recorded by others that no especial comment regarding this seem indicated.

In this series it will be noted there was not a single post-operative intraocular infection and no expulsive subchoroidal hemorrhage. Likewise, to date there has been no post-operative detachment of the retina. We have observed but one choroidal detachment, but patients are not examined with the ophthalmoscope until the fourteenth day, except in very rare instances.

COMMENTS

After the warning of such an outstanding authority as Colonel Wright,[4] one hesitates to make any dogmatic statements concerning an operation when the series reported is not large. When one has employed the extracapsular operation exclusively for nineteen years, however, and then adds the intracapsular procedure for a period of four years, it is difficult to avoid comparison.

One of the greatest advantages of the intracapsular operation is the freedom it permits in operating upon the immature cataract. Long waiting in semi-blindness can be avoided in most cases. The light nuclear sclerosis with clear peripheral cortex is ideal for the intracapsular procedure, and the operation can be undertaken as soon as the anterior chamber is of sufficient depth to permit a satisfactory section. The actual technic of the intracapsular procedure is simpler than that of the extracapsular, assuming one uses forceps for removal of the anterior capsule in the latter operation, since various manipulations incident to the removal of retained cortex, such as massage of the cornea and irrigation of the anterior chamber, are completely eliminated. Careless irrigation, with improper solutions, have not infrequently resulted in marked post-operative irritation and at times permanent clouding of the cornea.

Naturally one must practice the technic of intracapsular removal a sufficient number of times to become thoroly familiar with the operation. Coordination between pressure and light traction must be developed and fear of sudden vitreous loss must be overcome.

Fewer instruments are introduced into the anterior chamber and the incidence of infection should be lower. Usually the stay in the hospital has been definitely shortened. Post-operative complications have been reduced in this series compared with our extra-

capsular cases. The patients on the whole have been much happier due to the freedom from post-operative irritation and the shorter stays in the hospital. No after-cataract discissions are required with their attendant embarrassment.

I believe that in the average uncomplicated case the intracapsular operation, with some form of forceps extraction, should be the operation of choice. The optimum achievement in cataract extraction is obtained by a successful intracapsular procedure with the preservation of a round pupil. The feature of safety, however, must always be kept in mind and unquestionably some form of complete iridectomy adds to this, as stressed by Knapp and others. In old people, with their potential lack of co-operation and for whom a cosmetic result is not so essential, I believe iridectomy should always be used. In younger subjects, I prefer the simple extraction with peripheral buttonhole iridectomy. With a combined or preliminary iridectomy, as one prefers, the intracapsular operation appears equally as safe as the extracapsular procedure in the uncomplicated case. If one considers the possible post-operative complications of the capsulotomy operation, the intracapsular procedure, in my opinion, is the safer.

Peters,[7] in discussing Knapp's[7] paper on his third series of 100 intracapsular extractions, states that "if we eliminate the question of surgical skill and compare the results of the intra- and extracapsular methods, there can be no reason for difference of opinion as to the much greater value of the intracapsular extraction."

Knapp[7] points out that iris incarceration, vitreous opacities, after cataract, and glaucoma are all reduced to a minimum, while recovery is more rapid.

Torok,[9] in 1916, discussing his method of forceps extraction, states that it is very simple and easy for anyone who is experienced with the ordinary Von Graefe extraction and, further, that the danger of loss of vitreous is even less, because pressure is less when traction is used.

Contrary to the usual teaching and belief with regard to the greater hazard of vitreous loss in the intracapsular operation, I have grown less apprehensive about this phase of the procedure as the number of our operations have increased and, barring the exceptional case, it seems to me this complication should be no more frequently incurred than in the extracapsular method.

The outstanding features of the uncomplicated intracapsular extraction are the simplicity of the operative procedure, the very

slight post-operative reaction in most cases, the shortened post-operative healing period, and the rapid restoration of excellent vision.

Every ophthalmologist who is doing any amount of cataract surgery should be familiar with some form of intracapsular extraction. It may never completely replace the capsulotomy procedure, but certain types of cataract are best suited to the intracapsular method, and those who do not practice both methods will find themselves handicapped in the management of many of their cases.

BIBLIOGRAPHY

1. Davis, Fred'k A.: *Tribromethanol (Tribromethyl-Alcohol, Avertin) as an Anæsthetic in Eye Surgery.* Transactions of the American Ophthalmological Society, 1931.
2. Gradle, Harry: *Intracapsular versus Extracapsular Cataract Extraction.* Transactions Section on Ophthalmology, A.M.A., 1933.
3. Davis, Fred'k A.: *Capsule Forceps for Intracapsular Cataract Extraction.* Transactions of the American Ophthalmological Society, 1936.
4. Wright, Col.: American Journal of Ophthalmology, 20:376-387, April, 1937.
5. Vail, D. T.: *Hyphæma after Cataract Extraction.* Transactions American Ophthalmological Society, 1933.
6. Knapp, Arnold: *Complications of Forceps Intracapsular Cataract Operations.* Transactions American Ophthalmological Society, 1936.
7. Knapp, Arnold: Transactions of the College of Physicians, Philadelphia, 1932.
8. Lancaster, Walter B.: *The Cataract Operation.* Surgery, Gynecology, and Obstetrics, 52:452-463, Feb., 1931.
9. Torok, E.: Annals of Ophthalmology, 25:712, Oct., 1916.

DISCUSSION

Dr. John O. McReynolds, Dallas, Texas: The paper presented by Dr. Frederick A. Davis of Madison, Wisconsin, merits a close study and careful analysis. It is part of the systematic contributions on the subject of intracapsular cataract operations coming in now voluntarily from all portions of our country. These will doubtless continue to flow into our literature until we shall have, when they are all collected, a very valuable cross-section of American experiences and conclusions on this important and practical subject. We can then be in a position to blend these numerous individual results with the vast records of the men who have taken the initiative in stamping upon the profession indelibly the central principles and the favorite procedures that have given to their efforts "a local habitation and a name."

I must confess in this connection a little pardonable pride in the achievements of this gifted young surgeon, Dr. Davis, whose primary lessons in ophthalmology I had the honor to encourage and in some measure to direct. It gives me much pleasure at this time to assure our colleagues that whatever may be his methods and deductions, they will always be characterized by two features of the greatest value — clearness of presentation and honesty of purpose.

In a review of the multidinous records of cataract removal in the unbroken capsule, we must be impressed with the growing conviction throughout the profession that this is the end devoutly to be sought — if only it can be accomplished with the desired certainty and safety. In order that the lens may be delivered in its entirety, one essential requisite has always been to secure rupture of the zonule without rupture of the capsule. When this is done, the subsequent delivery of the lens can be easily and surely achieved.

Perhaps every ophthalmic surgeon has, at times, whether designedly or by accident, had the experience of removing the intact lens with an excellent result. It is, therefore, by no means a new or unusual occurrence. In the list of two hundred cataract operations originally presented by Jaques Daviel in his memorable paper in 1752 before the Academie Royale, there were many cases of intracapsular removal. It is a procedure then that dates back to the very dawn of this epoch-making advance in ophthalmic surgery. The primary step, the safe rupture of the zonule, was attempted always by one of three methods of producing a partial or complete dislocation of the lens. Of these three methods, one of them sought to break the zonule by pressure applied within the eyeball, either to the lens itself or directly to the zonule. Another of these procedures aimed to break the zonule by pressure applied to certain segments of the exterior of the globe. The third method, instead of resorting to pressure exerted either within or without the globe, had recourse to some form of traction applied to the capsule itself or to some portion of the mass of the lens. Thus we might say, that in all cases, the zonule was ruptured either by some form of push or else by some form of pull, or by varying combinations of these methods of force. At the present time, the fashion seems to be in favor of the combination of a push and a pull in varying degrees, according to the predilections or preference of the surgeon.

To illustrate, in the 1933 Transactions of the Section on Ophthalmology of the American Medical Association, our distinguished Dr. Harry Gradle of Chicago estimated that in his hands the element of push would be 80%, while the element of pull would be 20%. In the report of Dr. Frederick A. Davis before us today, his estimate is that in his hands, the push element would be 95%, while the pull element would be only 5%. Thus these elements vary in the experience of many very skillful operators from the 100% push of the brilliant Lieut. Col. Henry Smith of India, to the 100% pull by the versatile Professor Ignacio Barraquer of Barcelona.

From 1707, when Maitre-Jean, followed by Brisseau in 1709, gave the correct interpretation of the nature of cataract, down to the present time, there has been an almost unbroken interest in this surgical procedure. In 1707, you will recall that St. Yves removed three lenses in the capsule, and Pourfour du Petit in 1708 accomplished the same result. But in all of these cases, the lenses were already dislocated. As early as 1745 Daviel delivered

the intact lens by the aid of a spatula introduced within the eye, very much in accordance with the Smith procedure so widely employed at this time. This operation was followed by four other successful intracapsular operations, but it was not until 1750 that Daviel decided to remove all cataracts as completely as possible without couching or depression.

In the development of intracapsular operations we shall always be deeply indebted to the contributions by Alexander Pagenstecher and his brother, Herman, of Wiesbaden, who gave to the profession from their wide experience, covering 40 years after 1866, the most exact description of the cases suitable for intracapsular extraction. Herman Pagenstecher went further than most men of this day in his plan of operating on all favorable binocular cataracts; on one eye he used one method and on the other eye the other method, so that he had in these individual cases both the intracapsular and the capsulotomy method for comparison.

In addition to these discussions, numerous surgeons experimented with direct pressure within the eyeball upon the zonule. Especially prominent among these were Basso and Gradenigo of Italy, and Giles C. Savage and L. Webster Fox of our own country. Their instruments were variously called by the name of zonulotome, cataract detacher, and reclinateur, and have been warmly advocated by some but never generally adopted.

The real renaissance of intracapsular extraction, of course, began with Mulroney of Amritsar in the Punjab, the records of whom have been left to us through the writings of Mecher Chaud in 1894, followed by the marvelous record of Lieut. Col. Henry Smith in Jullender. The enormous volume of this concentrated work in India was crystallized most definitely by Col. Smith, but unfortunately his desire to bring the operation directly to the Western World was attended by many unfortunate results which would not, perhaps, have taken place in his usual theatre of operations in India.

In many of the operations, consisting of both push and pull, it is possible that associated with the grasping of the capsule there may be exerted a certain degree of push on the taut zonule, thus contributing both the benefits of the push and pull by the capsule forceps themselves.

In the meantime, we shall probably be wise in accepting the opinion written many years ago by good old Sir William Bowman of London to Edmond Landolt of Paris when he said, "I cannot doubt that the problem of what is to be accounted the best method in any class of cases will have to be worked out by our successors, with our minds remaining open to suit the thousand personal circumstances of our patients."

I am in perfect accord with the essayist with regard to the desirability and advisability of the intracapsular operation. There will be always many modifications in the technique employed. In my individual experience, I make practically all intraocular operations under an undivided conjunctival bridge which gives to me every possible advantage of a flap without involving the necessity of introducing sutures. In addition, it permits the immediate closure of the lids which is our best security against vitreous loss. It also shortens and renders more secure the process of healing with the consequent prevention of infection. It permits the patient a greater freedom of movement and reduces the period of confinement to the hospital.

I wish to congratulate our Dr. Davis not only on his operative work and its presentation, but also on the fact that he is making permanent his procedures by the use of moving picture illustrations which in time will give ophthalmologists a clearer idea of the surgical work that is being done throughout the world today.

DR. DERRICK VAIL, Cincinnati: More has been written about cataract, and the methods, maneuvers, and complications of its removal than on any other subject of ophthalmology. Whenever a group of ophthalmologists gather together, this subject is sure to come up and discussions and debates ensue, which would give the unknowing onlooker the impression that we are talking more and more about less and less. Why should there be the tremendous ingrained interest which this subject stirs in the heart of every ophthalmic surgeon? The answer, I believe, lies in the strictly personal makeup and skill of the operator as well as the thrilling drama of the task itself, a thrill comparable to that Little Jack Horner must have experienced with his Plum. At first glance we think that Dr. Davis' paper is just another one. Careful study and attention reveals a valuable contribution, in lucid style, which discloses a surprising amount of information in a relatively short space.

As a son of one of the American pioneers in intracapsular cataract surgery and nourished in my ophthalmic infancy on the advantages and perfection of the intracapsular operation, I am most of all impressed by Dr. Davis' metamorphosis from an old fashioned cataract surgeon of 19 years' experience to a modern one. The result of his recent work you have just heard. They speak for themselves. His statistics support unmistakingly the contention that a skilled ophthalmic surgeon, and one should not do any kind of ophthalmic surgery unless he is skilled, can in a relatively short time turn out intracapsular work as good and in many respects better than that done by his intracapsular predecessors.

There is no need to recapitulate the many advantages of intracapsular extractions. Dr. Davis has ably and fully covered this phase. Indeed to any observer, even casual, who has had an opportunity to compare the healing and final result between intra- and extracapsular cases, there can be no question. The ultimate masterpiece is the round pupil intracapsular cataract extraction, which, as Dr. Davis has pointed out, cannot always be done, considering factors of safety involved.

From my own experience, therefore, I heartily endorse, support and subscribe to every point Dr. Davis has discussed. These points are the importance of careful physical examination, the eradication of all focal infections, the preparation of the patient, Van Lint akinesia, bridle suture under the superior rectus muscle, some form of conjunctival or corneal suture, and in a certain percentage of cases particularly where the intraocular tension is upper normal, the retrobulbar injection of novocaine. The only objection to using it in all cases, in my hands, is the fact that the eyeball becomes too soft to allow effective and necessary counterpressure. Each of these procedures has demonstrated in dramatic fashion at some critical time or other its practical value. The main idea is to make them routine in all cases.

When it comes to the actual extraction of the lens, the individuality of each operator comes to the fore. It really doesn't matter what method is first learned and practiced. One should not remain stationary, but as confidence increases learn all methods. The versatile and resourceful surgeon can adjust the method to the eye while it is being operated upon.

The much, and I think unjustly, maligned Smith method serves its purpose on many an occasion. It is particularly valuable in the hypermature Morgagnian type as well as in the hard flat type of cataract. These, as Henry Smith has said, can be dislocated by an angry look. The experience in the Smith pressure gained by me in India twelve years ago has proved its worth time and again in expressing the lens dislocated previously with forceps. Indeed, in spite of the author's remarks on the subject, I am a little suspicious that he is using Smith pressure in his method. Smith pressure in India was less violent apparently than that used by its propounder on his tour in this country.

The Verhoeff method of grasping the lens at the equator, where the capsule is strongest, has been the only method, at times, by which I have succeeded in extracting the lens in its capsule.

A point lightly mentioned by the essayist deserves a little more emphasis. This is the study of the lens after extraction to see if there is any way we can recognize whether one way or another of operating is most likely to succeed. For several years we have been studying the anterior capsule carefully under the biomicroscope and recording our findings. So far, at any rate, there seems to be no relationship between the presence of shagreen, or thinness of the capsule, subcapsular vacuoles or water clefts and the tensile strength of the capsule. What to do with the green sticky immature cataract, fortunately rare, where the capsule ruptures before extraction, has been a puzzling problem. Irrigation does not help much and extraction of the rest of the capsule with forceps has only occasionally succeeded. There remain behind, then, fragments of sticky cortex and pieces of capsule which fill the entire pupil space in a few days. Subsequent needling often brings about a splendid result, but the disappointment remains.

The subject of vitreous loss can only be touched upon. Most surgeons agree that it is to be carefully avoided because late complications are almost sure to follow. I look with dismay on a cornea which wrinkles horizontally as soon as the section is made. It frequently presages vitreous loss. That vitreous loss is no greater after intracapsular than after extracapsular extraction is a fact attested to by many writers.

There are almost as many varieties of capsule forceps as there are of speculums. I don't think it makes very much difference whose type of forceps is used. The important point is that the edges must microscopically approximate and the heel must not close on the iris. The surgical supply houses have been very careless about these factors and often will sell a capsule forceps unworthy of the name. Examine your forceps with a magnifying glass, and if it is good, keep and preserve it as a priceless object.

In the study which I made on hyphema after cataract extraction, kindly mentioned by the author, the spontaneous or unexplained cases were regarded as caused by an increased intraocular tension, occurring in most instances from the third to the sixth day. This is probably the result of too

rapid and too tight closing of the wound and an overfilling of the anterior chamber. Wound reopening and hyphema were less often observed following the corneal type of incision than following the conjunctival flap and suture operation. It was concluded that this is probably due to delayed healing, which is common in the corneal section.

It was also concluded that the reason why hyphema occurs so seldom in the simple extraction is because, fundamentally, hyphema and iris prolapse have the same etiologic factor. Iris prolapse is notoriously frequent in simple extraction, and it may be that the prolapse obstructs the broken vessels of the wound and acts as a tampon.

Time prevents further discussion, which besides is unnecessary, because Dr. Davis, by his remarks, has held the mirror up to the experiences of those doing intracapsular work.

DR. E. C. ELLETT, Memphis: This paper opens a large field for discussion, and there are many matters of which one might speak at length in regard to his own experience. Of the many, I would like to speak of a few.

In regard to the capsule forceps, the Arruga forceps have proven most satisfactory. Before using these, I had used the Verhoeff forceps with great satisfaction, but the instrument as made by different manufacturers is not of uniform excellence, and a poor model will tear many capsules. The Arruga, or any forceps meant to grasp the capsule, must be kept in such condition that the whole of the two blades meet at the same time. If it grasps first at either toe or heel it will cut or tear many capsules.

As an element in satisfactory anesthesia, we are using retrobulbar injections with increasing satisfaction.

A very important matter, in my experience, is to have an ample corneal incision.

The author says that "true iritis was observed in only three cases." This claim is often made and probably a difference in experience may hinge upon what we mean by iritis, and where we draw the line. I must confess that the large majority of my cataract extractions show, at some time during the healing, what I take to be an iritis. I do not know what else will explain the ciliary injection that is present at some time in most cases, or the posterior synechia that will form in case there are capsular remains, of which, of course, there are none in a successful intracapsular operation. Another sign of iritis which may not be so well known or readily accepted as to its significance, is one to which I have called attention elsewhere, but which is not usually mentioned among the signs of iritis, though it is a sign upon which I rely very confidently as an indication of iritis. That is the appearance of grill-like figures in the cornea. Though some have interpreted them as folds in Descemet's membrane, which may explain some of the cases, the features I have in mind are not folds in Descemet's membrane, but are caused by dilated lymph channels in the substantia propria. They are quite a constant sign of iritis and consist of numerous translucent tracts or lines in the cornea, crossing each other at right angles, and located in the corneal tissue. They look exactly like the pictures showing the lymph spaces in the cornea, and they entirely disappear as the inflammatory action subsides.

Not so easily seen by oblique illumination, they are easily made out with the ophthalmoscope and of course with the slit lamp. However, we do not like to subject eyes freshly operated for cataract to the slit lamp. On the basis of these signs, most of the cases, both my own and others, that I have seen, show a certain amount of iritis after the extraction of a cataract, by whatever method it is done. I think there is no doubt, however, that all the inflammatory manifestations are milder and less constant after a well executed intracapsular extraction than after an extraction done by any other method.

A good practical way of estimating the incidence of iritis is to ask yourself in how many of your cases do you find that you have used atropine during the convalescence.

Dr. D. H. O'Rourke, Denver: It is not my desire to discuss the technical or the statistical details of Dr. Davis' paper, but I should like to emphasize a few points. Dr. Davis says, after nineteen years' experience with the extracapsular extractions, that four years ago he began doing intracapsular extractions in suitable and selected cases. The essayist is one of a very large group in this audience, and, I am sure, throughout the country at large, whose training was had before the days of intracapsular surgery. You well remember the serious discussions and advices pro and con in regard to this subject a few years back, particularly in regard to the unusual qualifications of those who should attempt this delicate procedure. This question is still an unsettled one in the minds of many, but should it be?

Dr. Davis has this to say: "The actual technic of the intracapsular procedure is simpler than that of the extracapsular, assuming one uses forceps for removal of the anterior capsule in the latter operation, since various manipulations incident to the removal of retained cortex, such as massage of the cornea and irrigation of the anterior chamber, are completely eliminated. Naturally, one must practice the technic of intracapsular removal a sufficient number of times to become thoroly familiar with the operation." Of course, it is understood that one should have sufficient experience with the capsulotomy method, and, to quote the author's last two sentences, because they appeal to me so forcibly in the light of my past and present experience:

"Every ophthalmologist who is doing any amount of cataract surgery should be familiar with some form of intracapsular extraction. It may never completely replace the capsulotomy procedure, but certain types of cataract are best suited to the intracapsular method, and those who do not practice both methods will find themselves handicapped in the management of many of their cases."

Dr. Davis in one paragraph very casually refers to capsular forceps of his own design. His characteristic modesty probably dictates such a casual reference. I have used his forceps a little over a year, and in my hands the instrument is better for the purpose for which it was designed than any I have used, and I recommend it for your consideration.

Dr. D. B. Kirby, New York: I believe that intracapsular extraction can be made as safe a procedure as extracapsular extraction. The cases should be selected. The intumescent types probably do better, and the heavily sclerosed lenses probably do as well with the extracapsular procedure.

In addition to the usual safety measures which have been advocated, we strongly urge the use of free external canthotomy whenever the ring of the lid margins with the speculum, or its substitutes in position, causes pressure on the equatorial position of the globe.

Two of the steps in the operation which I do are a little different from the average. After the section has been completed and sutures inserted, I use the point of a lens hook to break the zonular fibers in the lower semicircle by carefully applying light pressure directly over the zonular fibers—that is, just within the scleral ring at the limbus of the cornea. This is done before the lens capsule forceps are introduced, the object being to relax the capsule further and make it easier to pick up a fold of capsule and to shorten the period of use of the forceps within the eye. They are not applied until they are ready to assist in the delivery of the lens. The forceps may be applied below as in the Knapp procedure, and the lens tumbled, or above as Verhoeff has done, I use a cylindrical handled forceps with the Verhoeff stop.

The second feature is a suture guide in the central portion of the corneal flap. This permits careful retraction of the flap to afford a direct view of the base of the iris for the iridotomy. Also in the Verhoeff technic to avoid the need for complete iridectomy, I draw out the iris with the heel of the forceps, and with the aid of slight pressure below, which causes the lens to present in the wound, the forceps are applied directly to the anterior face of the equatorial region and the lens delivered through the round pupil.

Dr. Harry W. Woodruff, Joliet, Ill.: You will perhaps remember that in the discussion and in this paper there was considerable emphasis placed on push and pull. How true that is in life. You must have some of both, but as the old wise man, Socrates, said, "Nothing too much."

Dr. Davis spoke about the use of avertin, but he did not emphasize its use as much as I thought he would. Two years ago at our Academy meeting in Cincinnati, I read a paper on complications of the cataract operation, and I particularly mentioned avertin as an anesthetic in certain cases, cases of high blood pressure or cases of intraocular tension, or a combination of both, but avertin does two things: It reduces the general blood pressure and it reduces the intraocular tension very markedly. It seems to me as though you have a solution, for the time being at any rate, of the choroidal hemorrhage, expulsive hemorrhage, and in a measure vitreous prolapse. So I think that it would be a safety procedure in any of those cases of high blood pressure or high intraocular tension to use avertin.

Cataract operators are still using the speculum. Dr. Fisher, of this city, has advocated the use of a lid retractor for a long time, as well as the intracapsular operation. I think that cases of vitreous prolapse can be avoided much more easily if the lid is held by a retractor rather than if the lids are kept apart by a speculum.

There is one more point, and that pertains to the use of sutures in the lid. By the use of two sutures in the upper lid, held so that the upper lid can be controlled by these sutures, if there is any sign of vitreous prolapse one can immediately close the eye, bringing the lid down over the cornea with these sutures.

I think that it is very creditable to our profession, our specialty, that advances are being made, and I think it is worth while for you to give these particular items very close attention. I know it is difficult to change habit. If you are in the habit of doing a thing a certain way and you have been successful in doing it, it is very hard to change, and I regret exceedingly that I am in that class, but these changes I have mentioned I am satisfied are well worth while.

DR. LUTHER C. PETER, Philadelphia: I heartily endorse what Dr. Woodruff has said. The two main points which he offered are the ones I desire to stress. I refer particularly to the lid sutures and the use of the lid retractor rather than the use of the speculum. Intracapsular extraction is no longer in its experimental stage, but is the operation of choice in most cases of several varieties of cataracts in the adult. In this group, of course, traumatic cataract is the exception, and in the interest of better surgery, therefore, it is timely to discuss points in the technic in which individual experiences may help others to increase their percentage of good results.

My discussion will be limited to two phasees of the work. The first is that of anesthesia. If the patient is cooperative good local anesthesia may be adequate to yield excellent results. In my experience, comparatively few patients are capable of perfect cooperation even though they try to the best of their ability. A bridle suture placed under the superior rectus is of great advantage to those operated upon under local anesthesia, and is a routine procedure in my work whether I operate under local or under avertin anesthesia. In a majority of cases, avertin is used as the safest method to obtain perfect results. When properly administered, few cases are nauseated.

Vinethene is a rather effervescent anesthesia. It does not produce nausea except in a few individuals. A very, very small amount is needed, and the patient recovers very quickly. I think it is ideal to reinforce avertin whether in cataract surgery or any other form of surgery. The patient, by the use of avertin, is spared the dread of the operating room. He is unconscious of having been moved from his bed to the operating room. Recovery from the anesthetic as a rule is uneventful and not disagreeable, and the satisfaction with which the surgeon can proceed uninterruptedly with the delicate manipulations required of the intracapsular surgery is of maximum importance.

In addition, of course, (Dr. Woodruff has expressed this point) it reduces the blood pressure and the intraocular pressure and therefore commends itself. There are a few contraindications for its use.

The second point in the procedure is the grasp of the forceps and manipulation of the same in the extraction of the lens. The capsule forceps, of which Arruga's model is the best type, should grasp the capsule close to

the lower border of the lens, under the iris, even in a well dilated pupil. At this point in the periphery, the capsule is dense by reason of reinforcement from the zonular fibers. Rupture of the capsule is less likely to occur, first, because of this increased density; secondly, the bite of the capsule at this peripheral point does not place the entire capsule under tension, as when the grasp is taken near the center of the lens, and, thirdly, rocking of the lens in a circumferential direction can be accomplished with a minimum of violence and yet with effective results.

In my efforts to reduce the number of ruptured capsules, this procedure has proven to be the greatest factor. A large corneal section is a prerequisite to success, but without a peripheral bite by the forceps, delivery in capsule may fail even if the section is generous. The one advantage of the Verhoeff method of extraction with Verhoeff's forceps is the point which I have discussed. In his method, however, the necessity for an iridectomy, the inevitable presentation of the upper edge of the lens first, rather than the lower edge by tumbling and the breaking of the zonular fibers above, at the beginning rather than at the end of the delivery, are objectionable features for an ideal operation.

In the method which I employ, tumbling of the lens is a natural consequence and vitreous loss is not apt to occur, first, because the iris sphincter is intact; secondly, because the corneal wound is closed by the body of the lens; and, thirdly, because the last zonular fibers to rupture are those from above, as the lens is delivered in capsule by a sweep of the curve of the Smith hook.

In relation to the push and pull phenomena to which Dr. Davis has aptly referred, my technic is a bit reversed. I depend more upon the circumferential rotation in the peripheral bite to dislocate the lens than upon the Smith local, and I depend upon the Smith local largely at the end of the operation and the curved part of it to break the upper zonular fibers, but, as others have pointed out, this is a matter of individual technic, and we learn day by day and we shift our position day by day in some of the refined technic of this work.

There are other phases of intracapsular extraction which might be discussed to advantage, but the two points to which I have confined my discussion are of greatest importance in securing a high percentage of successful deliveries, a percentage which should run close to or better than 90 per cent. It is a great source of satisfaction to find so many operators turning from the old two- and even three-stage operation, to one which subjects the patient to but one surgical procedure, with rapid convalescence, better central visual acuity, and if the pupil is allowed to remain intact, vision which is most comfortable because of the absence of light glare.

Dr. W. A. Fisher, Chicago: Dr. Davis is certainly to be congratulated on the results of 200 intracapsular operations by any method, whether it is forceps or otherwise. It is needless for me to say that we all know that

when one can get the results that Dr. Davis has recorded, he is simply a first-class operator.

It has been stated that this is a very simple thing to do, that the intracapsular operation is a very simple procedure. I think it is about time that someone tells you how to make this simple. I can imagine that there is a large number of members of this society who believe that any cataract operation by any method is a very simple procedure; then there is another crowd who do not believe it is so simple, and I want to speak of the other crowd.

This can be made very simple. I have stated this in a paper before and I just want to emphasize it, that the crystalline lens of a freshly killed chicken placed in a mask made with an ordinary spool is used to acquire this technic, and a Hildreth lamp makes the practice more simple, and if the Hildreth lamp could be added to Dr. Davis' technic, I feel that it would be perfect.

DR. FREDERICK A. DAVIS, closing: When I was invited to read a paper before this meeting of the society, I submitted the title of this paper, and received a gracious reply from one of my good friends with the request that some other subject be chosen, since he thought that cataract had been so frequently discussed in medical meetings it had lost its lure. I am very happy to see from the number of discussions and the length of them that it still holds its interest; in fact, I believe that when this is lost our branch of surgery will cease to exist as a specialty.

In regard to one or two remarks made by the gentlemen who were kind enough to discuss the paper, I appreciate very much the message from my old teacher, Dr. McReynolds, from whom I received my inspiration to enter the field of ophthalmology. The impression I received of his brilliant cataract surgery in my early days as a young man remains to this day.

In regard to Dr. Woodruff's remarks and those of Dr. Peter, also, concerning avertin, as I mentioned in the paper, avertin was used in 18 per cent of the cases. I have published two previous communications on the use of avertin and I did not wish to burden the audience and the paper with too lengthy a reference to this work. Since it has been brought up, however, I shall say one word further: I brought out in my original studies on avertin the great advantage of its use in operating upon cases with increased intraocular tension, as it frequently reduces the tension very markedly.

In regard to the speculum mentioned by Dr. Fisher, I realized that I probably would be criticized by most men because I used the speculum thruout the operation. I have used hooks and I know the teaching with regard to the use of lid hooks. However, I have taken up the intracapsular operation since the days of akinesia. It seems to me that makes a great difference. We simply do not need hooks as we used to in the days when patients were squeezers. They rarely squeeze after thorough and complete injection of the orbicularis.

I often find I am tempted to remove a speculum in conformity with my early instruction. I may say that my original teaching compelled me to remove the speculum immediately after the section. That is what I did for fifteen or twenty years, but I do not do that in this hazardous (?) intracapsular operation. Why? Because I have learned to use novocaine injection of the orbicularis. Undoubtedly, I may meet with accidents because of this sometime, but I feel that manipulation with the forceps and pulling on the hooks up and down by an inexperienced assistant is probably more hazardous than leaving a light speculum in place, which can be quickly and easily removed, such as the Skeel modification of the Weeks.

In closing, I deeply appreciate the generous criticism of the various gentlemen who have been kind enough to discuss the paper.

OCULAR MANIFESTATIONS OF ENDOCRINE DISTURBANCE

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ALTHOUGH much is known about the functions of the endocrine glands, their study still remains the most fertile field for advancement in medicine. The actions of these glands are so greatly influenced by environment, vitamins, chemicals and the emotional states that animal experimentation is not conclusive. The action of one gland is so intimately bound up with those of all the other glands that it is almost impossible to isolate the hyper- or hypofunction of any single gland. Other glands, in an effort to make up the deficiency of a hypofunctioning gland, may develop hyperfunctions of their primary secretions or their component glands. Then the vitamins, which are to plant life what the hormones are to animal life, are essential to the proper functioning of the endocrine system. Finally, biochemical reactions and the nervous system regulate the endocrine function and the endocrine function controls the biochemical process and the nervous system.

Because of this complexity and the interrelation of the various endocrine glands, nervous system, and biochemistry, it can readily be seen that, after all, the clinical symptoms and physical characteristics render the most reliable information applicable to clinical medicine.

Because of the facility of studying directly under magnification the living ectoderm, mesoderm and to a lesser degree the entoderm, and because of the dual nerve control system of the vagus and sympathetic, the eye provides an excellent medium for the study of endocrine disturbances.

Since we have such a field for the study of endocrine disturbances it behooves us as ophthalmologists to train ourselves to recognize the various signs of endocrine malfunction in order better to qualify ourselves as consultants and practitioners of ophthalmology.

No effort will be made to review the extensive literature, as that has been done quite completely by Von Szily[1,2] and Velhagen.[3]

The autonomic nervous system is so intimately associated with the function of the endocrine system and the eye that a brief review is essential to a clear understanding of the action of the endocrine glands in relation to the eyes. The autonomic nervous system is not an independently controlled nervous system, but the involuntary outflow from the cerebrospinal nervous system with peripheral plexi at the sites of distribution which are connected by neurones to the cerebrospinal system. These are subdivided into the sympathetic, parasympathetic and visceral systems. The sympathetic comprises the thoraco-lumbar outflow, while the parasympathetic includes the cranial and sacral outflows.

In hyperstimulation of the parasympathetic (vagotonia), there is increased salivation, perspiration and lacrimation, dermal and vasomotor changes and motor reactions manifested in contraction of the pupils, accommodative spasm, respiratory contraction, and spastic colon and anus tenesmus, while in hyperstimulation of the sympathetic there is diminished lacrimation, perspiration, and salivary secretion, the pupil is dilated, the pulse rate is increased and there is colonic-rectal atony. In other words, the sympathetic and parasympathetic systems, when supplying the same organ, act antagonistically. There are some organs supplied only by the sympathetic and in these the sympathetic has an added mild parasympathetic action.

The adrenal medulla, anterior pituitary, thyroid and gonads stimulate the sympathetic, while the posterior pituitary and the adrenal cortex stimulate the parasympathetic. In pilocarpine we have a drug that stimulates the vagus system, while atropine paralyzes its terminals.

Before attempting to attribute any ocular manifestation to endocrine disturbance, other disease entities should be sought for and eliminated because in the presence of any other disease one is not justified in drawing a definite conclusion of endocrine pathology.

The blood sugar, non-protein nitrogen, urea, calcium, uric acid, cholesterol and chlorides are of value in arriving at a conclusion. For the blood sugar is affected by the pancreas, thyroid, suprarenal and pituitary; non-protein nitrogens, urea, uric acid and creatinin by the supra-renals, thyroid and pancreas; calcium by the parathyroids; cholesterol by the thyroid.

THYROID

Of all the endocrine glands, the function of the thyroid is best known and apparently most frequently affects the eyes, either because its dysfunction is more frequently recognized or because it has a greater influence on the appearance of the eye.

The internal secretion of the thyroid assists in the stimulation of the sympathetic nervous system and mentality and acts as a catalytic substance, expediting metabolic processes and facilitating chemical changes in all the cells of the body, especially reactions involving iodine, calcium, sodium chloride and phosphorus. It influences the function of the pancreas, pituitary and gonads.

As is well known, the basal metabolic rate is unreliable unless several tests be made. The blood cholesterol is perhaps more reliable, being low in hyperthyroidism and high in hypothyroid function. However, the blood cholesterol is also increased by an excess of vitamin D and lowered by the deficiency of vitamin D. Because of this unreliability of laboratory procedures, recognition of the ocular signs of thyroid dysfunction is of great importance in arriving at a diagnosis.

The most striking ocular sign of hyperthyroidism is exophthalmos, usually binocular. This is due to a round-cell infiltration and edema of the muscles and orbital tissues. In some cases the exophthalmos is aggravated by thyroidectomy. These cases seem to be due to a hyperadrenalism and Crile has relieved many of them by denervation of the supra-renal glands. If the exophthalmos is not relieved, lagophthalmic keratitis may develop, resulting in very troublesome ulcers or loss of the eyes.

There are other ocular signs associated with hyperthyroidism, alone or in conjunction with exophthalmos: Graefe's sign, failure of the lids to follow the downward motion of the globe; Stellwag's sign, diminution in the frequency of blinking; Dalrymple's sign, deficiency of convergence; and Gifford's sign, a transverse ridge on eversion of the upper lid due to spasm of the orbicularis. These signs are all the result of a hyperstimulation of the sympathetic. There may also be muscular paralysis or heterophoria as the result of pressure, and dilated pupils due to sympathetic hyperstimulation.

In hypothyroidism, the skin of the lids is edematous, the brow hairs and lashes are sparse and brittle and there may be marginal blepharitis, eczema and chronic conjunctivitis. Enophthalmos may be present. Occasional cases of nystagmus have been ascribed to

hypothyroidism. In marked cases there is a yellowish fold of edematous conjunctiva in the lower cul-de-sac. Lacrimal secretion is diminished and the frequency of blinking and squinting is increased, in some cases very markedly. Luetic interstitial keratitis occurs much more frequently in hypothyroid patients and the excellent and rapid results obtained in these cases by fever therapy is most likely due to stimulation of the thyroid and supra-renals, which in turn stimulate the reticulo-endothelial cells. Myopia and opacities of the lens are frequently associated with hypothyroidism. Multiple non-infected chalazia are frequently the result of hypothyroidism. I have found the incidence of hypothyroidism high in cortical cataracts. Strabismus has frequently been found associated with hypothyroidism.

I have found that hypothyroidism may easily be overlooked in the children of the more prosperous classes. It should always be suspected in children with chronic eczema and catarrh.

I have associated with hypothyroidism a sign not previously published, viz.: an edema of the retina around the disc and macula extending into the retina as much as 4 or 5 disc diameters. I have found the disc elevated as much as 3 diopters and the vision reduced to 20/40 by this edema. At first, this condition was attributed to allergy, but in a number of these cases it was not possible to demonstrate an allergy of any known allergen. Further investigation demonstrated deficient thyroid function in most of these non-allergic patients. Then I was able to demonstrate deficiency of thyroid function in most of those in whom allergy was present.

The only reference found relative to retinal changes in hypothyroidism was by Englebach,[4] who states, "The retinal signs present in some cases of myxedema are often thought to be albuminuric retinitis." That represents the condition found in extreme cases but in patients with mild edema of the retina the existence of a low-grade hypothyroidism can usually be confirmed by several basal metabolic tests, the blood cholesterol and the therapeutic test.

PARATHYROIDS

The parathyroids control the calcium-phosphorus metabolism and assimilation which acts as a sedative to the irritating effects of sodium on the nerves of the voluntary and involuntary muscles. Consequently, with deficiency of the parathyroid secretion, one develops symptoms of calcium deficiency, that is, increased muscular rigidity with fine tremors of the voluntary and involuntary

muscles. This calcium deficiency has been termed "diabetes calcareous." It has been suggested that vitamin D may take part in the function of the parathyroid.

I have observed no ocular manifestations of hyperparathyroidism. Lack of parathyroid secretion disturbs calcium metabolism, resulting in a vagotonic activation of muscle tissues and may be reflected in clinical ophthalmology as ciliary spasm, blepharospasm and idiopathic spastic entropion, twitching movements of the lids and eyeballs and hypersecretion of tears. The calcium disturbance may produce zonular cataract. There is a close association between cataract and parathyroid tetany in children. I have seen cases of strabismus without refractive errors develop during convulsions, which most likely were the results of acute hypoparathyroidism, since 90% of convulsions in children before 2 years of age are due to this condition.

PITUITARY

The anterior lobe of the pituitary regulates growth and development and has a great influence on the gonads, mammæ, thyroid and adrenals. It is a sympathetic stimulator and assists in the metabolism of protein, fats, water, calcium, etc. After removal of the anterior lobe, the adrenal cortex and gonads atrophy. Part of its action on the eye is through its effects on the sympathetic and parasympathetic nervous systems.

Opacities of the lens are seen in hyperfunction, as in acromegaly. In pregnancy there is a physiologic enlargement of the pituitary causing a bitemporal contraction of the visual field. In marked enlargement of the pituitary the fields usually show bitemporal hemianopsia and occasionally atypical defects. Contrary to general belief, pathologic pituitary enlargement does not always produce a bitemporal contraction, but one or both fields may be concentrically contracted, or, if only the posterior lobe is enlarged, one of the optic tracts may be involved with the production of a homonymous hemianopsia. These field changes are associated with blurred vision or amaurosis, with swelling of the optic disc and eventually secondary optic atrophy. With most of these enlargements there is more or less headache. In tower-skull, which usually is associated with hyperpituitarism, the orbit and optic foramen are contracted. In minor disturbances there may be transitory visual field disturbance and scintillating scotoma.

Anterior pituitary hypofunction has been suggested as a cause of retinitis pigmentosa. Thirty-five cases have been reported as Laurence-Beidl syndrome. I have seen a brother and sister with this syndrome, which cases have not been reported. They have a Fröhlich type of obesity, deformity of the skull, retarded mental development and retinitis pigmentosa. These cases are usually also hypothyroid. The use of anterior pituitary extract in conjunction with thyroid has been suggested in the treatment of cretinism, because of the frequent finding of a small sella in the cretins.

The function of the posterior pituitary is that of stimulating the sympathetic and involuntary muscles. It influences the metabolism of sodium chloride, water and carbohydrates. In hyperfunction it produces a sympathetic hyperstimulation, increases pigmentation and increases smooth muscle tone. Its secretion is a pressor principle more persistent than epinephrin and acting synergistically with it. It is an antidiuretic, as in diabetes insipidus.

Angioneurotic edema is sometimes associated with dysfunction. Lenticular opacities associated with diabetes insipidus are sometimes found. Night blindness may result from hypofunction. These patients have heavy superciliary ridges, shaggy eyebrows and thick lashes and, frequently, blepharitis. Mild ptosis may be a result of a posterior lobe hypopituitarism.

In combined dysfunction of the pituitary and gonads are found seborrhea and acne that may involve all ectodermal structures such as the skin, conjunctiva, cornea and lens. Leber's disease (hereditary optic atrophy) has been placed by some observers in the category of pituitary disturbances. Zentmayer noted definite changes in the sella in a number of cases and restoration of vision has been reported following the use of pituitary therapy.

ADRENALS

The adrenal glands are essential to animal life. The glands are made up of two parts, the cortex and medulla, each having a distinct action. The cortex is the part of the gland indispensable to life.

The cortex assists in regulating the sodium and potassium concentration of the body, controls pigmentation, and is a parasympathetic stimulant. It neutralizes toxins. It assists in the assimilation of vitamin C (cevitamic acid) and in that way should act favorably on cataracts due to vitamin C deficiency. With deficiency of the cortex there is loss of sodium and a retention of potassium

thereby causing an alteration of sodium potassium ratio. This is followed by loss of water and loss of weight, which results in increase of the non-protein nitrogen and urea content of the blood. Diminution of the cortex results in a lowering of the blood sugar and glycogen stores and in an increased sensitivity to insulin. In marked adrenal cortex deficiency the skin becomes irregularly pigmented and darkened as a result of dehydration. There occurs also diminished oxygen consumption, muscle weakness, increased capillary permeability and in extreme cases circulatory collapse resembling histamine shock.

Cortical hypoadrenalism may result in enophthalmos and occasionally tumors and hyperplasia of the adrenal cortex are causes of exophthalmos.

The medulla of the adrenal affects the pancreas and apparently its secretion is antagonistic to insulin, thereby increasing the blood sugar and it may cause sugar to appear in the urine. It stimulates the thyroid function and their combined actions regulate the body temperature and metabolism. The action of adrenal medulla is pressor, its effect being more rapid but of shorter duration than the posterior pituitary secretions. It is a definite sympathetic stimulator and causes contraction of the smooth muscles, thereby affecting muscle tone and vasomotor control.

Myopia and some cases of glaucoma have been attributed by some to adrenal deficiency.

PANCREAS

The internal secretion of the pancreas controls carbohydrate metabolism. The external secretion controls digestion of fats and regulates the alkaline reserve.

In hypo-insulinism there is increased blood sugar, resulting in heightened susceptibility to pyogenic infections. By sudden lowering of the blood sugar there may be produced swelling of the lens with rapid refractive changes. Previously existing lenticular opacities may progress to maturity or opacities may form in a previously clear lens. Uveitis may be caused by hypoinsulinism. There may be retinal hemorrhages or even massive hemorrhages into the vitreous as a result of changes in capillary permeability. However, hemorrhages in the retinæ of these cases are most commonly due to vascular changes, the result of prolonged increased blood sugar. Retinal lipemia may also be a result of hyperglycemia.

GONADS

The gonads are not self-regulating organs that control their own function. The anterior lobe of the pituitary gland is the main regulator of the sex gland activity. The supra-renal cortex is also a gonadal stimulant. The thyroid, either through its interrelation with the pituitary and supra-renal cortex or direct action on the gonad, also is a factor in gonadal stimulation, but this function is not well understood.

The gonads are a sympathetic stimulant. As a result, patients with deficient gonadal hormones exhibit symptoms of hyperparasympathetic stimulation as manifested by vagotonia.

I have had a number of patients with hypogonadism with definite signs of hyperparasympathetic stimulation or vagotonia. Most of these patients had marked discomfort in the eye with associated rather constant headaches and inability to use the eyes without discomfort and complained of blurred vision. No abnormalities of the eyes could be found. Many of them manifested symptoms of sinus disease including enlargement of the blind spot. Most of these patients had consulted several reliable oculists previously with only temporary or no relief from the symptoms. Many of these patients were relieved with antuitrin, even castrated women responding to the treatment, and in one case the blind spot enlargement returned to normal in ten days. Of all the procedures I have used in ophthalmology this is one of the most appreciated by the patients.

I have had a few males with hemorrhages in the retina and vitreous who gave positive tourniquet tests and whose condition did not respond to cevitamic acid, although no other cause could be found to account for the hemorrhages, who immediately responded to whole ovarian substance and the tourniquet test became negative. After discontinuing ovarian extract the hemorrhages returned and the tourniquet test again became positive. These patients I have classed as mild hemophilia, for which the treatment is whole ovarian substance.

DISCUSSION

In the literature many ocular diseases have been found attributed to endocrine disturbances. Glaucoma has frequently been mentioned as being of endocrine origin. There are so many types of glaucoma that it could not be expected to be the result of a single endocrine syndrome. Cases have been reported as due to hyper-

and hypothyroidism, hyper- and hypopituitarism, allergy due to spleen deficiency, cortical hypo-adrenalinism and gonad deficiency. I have seen several patients in whom glaucoma was apparently due to endocrine dysfunction, but none of them were due to a single gland and no two cases were identical. I have used supra-renal cortex with spectacular drop of intraocular tension after the first injection but with no results upon subsequent injections. Likewise, I have used tissue extract with no definite results.

Some cataracts have been placed in the category of endocrine disturbances by a number of authors and cases have been attributed to hypofunction of the pancreas, parathyroid, thyroid and gonad, supra-renal cortex and hyperpituitary, both anterior and posterior lobes. I have seen patients with cataracts with associated dysfunction of all the glands mentioned above but the one finding most frequently encountered in cataract patients (both congenital and senile cortical types) is hypothyroidism, and in a great number of these hypothyroids there is also an associated hypogonadism.

Myopia is frequently ushered in by spasm of accommodation and can frequently be greatly diminished by atropine. Physiologically a spasm of accommodation should be due to hyperparasympathetic stimulation or possibly to a hyposympathetic stimulation. On a physiological basis, cases of acquired myopia may possibly be due to endocrine disturbances, either cortical hyperadrenalinism lar hypoadrenalism, hypothyroidism, hypogonadism and hypoparathyroidism.

Keratoconus has also been reported by several authors as being endocrine in origin. Some feel it to be due to thyroid deficiency and others as being polyglandular in origin. Others consider it due to calcium deficiency, probably parathyroid in origin. The findings of the above were not conclusive. However, I feel that there probably is a relation to some endocrine dysfunction not yet fully understood.

Some have associated trachoma with status thymolymphaticus, pituitary deficiency, and the adenoid type. Their findings were not convincing.

Some cases of uveitis have been associated with endocrine disturbance, but, excepting uveitis due to diabetes, most of them were only suggestive of an endocrine dysfunction.

Retinitis pigmentosa, besides those cases mentioned above, has been associated with dysfunction of most of the endocrine glands,

but on a physiological basis it is not likely to be associated primarily with any gland but the pituitary or adrenal.

In fact, some authors have attempted to associate practically every clinical entity in ophthalmology with certain endocrine dysfunctions, most of which contentions have very little merit.

I do not wish to convey the impression that endocrine therapy is essential in all conditions that are enumerated as being endocrine in origin. It is well realized what the proper emotional environment will do to endocrine imbalance. Likewise, a vacation to the country will frequently readjust a marked endocrine imbalance. However, there are so many patients who cannot be sent to the country and whose environment cannot be readjusted, that it becomes necessary to treat such patients as well as some dysfunctions that are primary in nature and in whom endocrine therapy is essential.

CONCLUSION

In conclusion, I wish to emphasize that the eye is the most fertile field for the study of endocrine disturbance and that it is the duty of every ophthalmologist to study the numerous manifestations of the various endocrine disturbances in order better to qualify ourselves as consultants.

Many of our heretofore hopeless conditions are being solved by endocrine studies and may we look forward to the day when many progressive conditions will be arrested by endocrine therapy.

BIBLIOGRAPHY

1. Von Szily, A.: *On the Pathological Relation of the Visual Organs to Endocrine System,* Zent. f. d. ges. Ophth. 33:161, 1935.
2. Von Szily, A.: *About Different Eye Diseases Which Are Thought to Be Related to Endocrine Disease.* Zent. f. d. ges. Ophth. 34:193, 1935.
3. Velhagen, K., Jr.: *Observations on Disturbances of the Endocrine System and the Eye.* Klin. Monatsbl. f. Augenh. 96:577, 1936.
   Von Szily, A.: *Disturbances of the Endocrine Secretions and Their Effects on the Eye.* Zent. f. d. ges. Ophth. 32:97, 1934.
4. Engelbach, Wm.: *Endocrine Medicine.* Vol. 3, p. 325.

DISCUSSION

DR. A. D. RUEDEMANN, Cleveland: First, I wish to thank Dr. Lemoine for asking me to discuss this paper on endocrine disturbances. My own experience with endocrine disturbances has been rather unsatisfactory, for the simple reason that having made a diagnosis, very little advice in the way of treatment is available. I might advise one thing, when you complete a diagnosis, leave the door open a little so you can write in the second diagnosis, for in most instances it is rather difficult to substantiate the first diagnosis.

As you all know, there are very few accurate tests for endocrine dysfunction. In patients with hyperthyroidism, the one field I think that offers the most as far as the ophthalmologist is concerned, we see few of the patients until it is too late to offer them much as far as the eye is concerned. As soon as the patient develops a rapid pulse, a high basal rate and a retraction of the lids, he is rushed to the hospital and his thyroid removed. This is a mistake for two possible reasons: Many of these people do not have hyperthyroidism, and most of them do not have an exophthalmos. I believe that many of these cases are a polyglandular disease, and by saying that, you are merely covering a multitude of sins, because I do not know what it means myself, but it is a disturbance of the entire glandular system, and the thyroid is merely one link in the chain of glandular disturbance.

There is one field, however, where the ophthalmologist should be concerned, and that is in the acute hyperthyroids that are present in adults. Hyperthyroidism coming on in adults is a field of diagnosis for the oculist and the oculist usually misses it. In a group of patients which we studied, 75 per cent who had acute hyperthyroidism with progressive exophthalmos had been given glasses during the acute phase of their disease and during the progression of their exophthalmos. A taking of the pulse rate, or merely measuring the exophthalmos with a Hürthle's exophthalmometer would have shown the operator the exophthalmos and that the difficulty with the eyes was due to thyroid disease. Such cases should be regulated by the oculist and not by the surgeon.

Many of these cases, if they were not operated upon and were put to bed, would lose their high basal rate and would lose their widening of the palpebral fissure, and would not develop the malignant exophthalmos which often follows removal of the thyroid.

In the group labelled hypothyroidism, my experience has not been that of Dr. Lemoine. I have been unable to find any enophthalmos associated with hypothyroidism. There is, however, another group of patients which I think is clinically important to us who have a weakness of accommodation, a so-called pseudo-Parkinsonian syndrome. They fatigue very rapidly on using their eyes for close work. They fatigue toward ten or eleven o'clock in the morning, and they have some susceptible head pain due to the using of the muscle in offsetting the accommodation convergence fatigue. Small amounts of thyroid extract should help them very materially, but it should be given by one who knows the glandular therapy.

It is unwise for the ophthalmologist to handle glandular dysfunction therapeutically. If you prescribe thyroid extract, you must be ready at any

moment to stop the treatment and subject the patient to hospitalization to reduce the hyperactivity of the thyroid gland.

Most of the parathyroids we have seen have been surgically positive parathyroids. It is where, in removing the thyroid gland, the surgeon has inadvertently removed the parathyroid and the patient developed a parathyroid tetany. The only thing to remember in hypoparathyroids is that any case that has had thyroid surgery may have some tetany, and unless you know the calcium level before you attempt the surgical procedure on such a patient you may regret the action.

I have had the sad experience of operating on a doctor's mother who said that she had had a thyroid operation and he knew that she did not have tetany, but immediately after her thyroid operation she developed a cyclonic type of convulsion and she evulged most of the iris and vitreous. One does not forget such an experience.

There is an increasing group of patients who, I think, have had a hypoparathyroidism without a surgical history, who have a pseudo-tetany. They must be treated with gland dicalcic phosphate, and again it belongs in the field of the endocrinologist.

I have been unable to attach anything to the pituitary gland or the adrenals.

In the group of the pancreas, Dr. Lemoine has covered the subject very well, but in patients with hypo-insulinism, there can be a group which does have associated chronic ocular fatigue. They are a group which is apt to have ocular muscle imbalance as a part of the fatigue. Their entire body musculature is low, and if they are given extra sugar or some insulin to increase their appetite, they will be able to carry on.

In closing, I offer a word of warning in treating cases with endocrine disturbance, that it is not a field for the ophthalmologist.

Dr. Charles A. Bahn, New Orleans: Dr. Lemoine is the first ophthalmologist, to my knowledge, who has written on the important chemical and physiologic interrelationships which exist between Vitamins and Hormones and which apparently marks a step forward in endocrinology. Thus, Vitamin A deficiency frequently causes night blindness in man or animal, and the pituitary melanophor hormone is reported to cause night blindness in animals. Vitamin C, cevitamic acid, and an adrenal cortex hormone are closely associated chemically and seem to be a contributing cause in some forms of cataract. Vitamin D and Parathormone both affect calcium metabolism and apparently are a causative factor of other forms of cataract in man or animals. These illustrations suggest that both vegetable and animal catalytic agents together play a more prominent part in the chemistry of health and disease than has previously been thought and that their interaction is a promising field for investigation.

As our knowledge increases, we are becoming less dogmatic about the role that specific gland dysfunctions play in the causation, aggravation or association of ocular symptoms. The following etiologic classification has been of service in my instruction courses during the past several years.

1. Ocular symptoms caused by endocrine dysfunction;
2. Ocular symptoms aggravated by endocrine dysfunction;
3. Ocular symptoms associated with endocrine dysfunction, the causative relationship as yet undetermined.

The first group is illustrated by lid bronzing, more or less characteristic opacities in cornea and lens, uveitis, retinitis and changes of refraction in pancreatic diabetes; by lid pigmentation in Addison's disease; by perinuclear opacities in hypoparathyroidism; and by visual disturbances, with or without inflammation, involving the retina-optic nerve tract in some pituitary lesions.

The second group is illustrated by the increased severity of, especially, uveal and retina-optic nerve disease of different forms during menstruation, pregnancy and menopause; by the increased frequency of interstitial keratitis in hypothyroid congenital luetics and the increase of lid pyoderma during adolescence.

The third group is illustrated by the association of retinitis pigmentosa with polydactilism, obesity, genital underdevelopment and psychic changes which together form the Laurence-Moon-Beidl syndrome. In this syndrome a form of congenital pituitary dysfunction is apparently involved. Another illustration is the association of conical cornea with the Fröhlich type of pituitary dysfunction; angioneurotic edema with adrenal insufficiency; blepharitis and premature presbyopia with hypothyroidism; and numerous congenital ocular defects with thyroid and other transmissible endocrinopathies.

Whether or not you accept the above illustrations in the order in which I have placed them is of minor importance. More important is that we learn to think of ocular endocrine symptoms in terms of causation, aggravation or association.

The controversial side of ophthalmic endocrinology largely centers around the existence of a specific toxicity of the ductless glands for the ocular tissues. The frequent and rather characteristic involvement of skin, lens and uveal tract in pancreatic diabetes strongly suggests that endocrinopathies alone may cause ocular disease independently of autonomic nerve imbalance such as occurs in hyperthyroidism and pressure reactions in pituitary tumors. Although a review of the literature convinces me that specific ocular toxicity does rarely exist in some persons with thyroid, parathyroid, pituitary and other endocrinopathies, many ophthalmologists do not concur in this opinion, which illustrates the necessity of more observations by more observers.

Of what service is endocrinology to you in everyday practice? Without it, you could not fully understand the ocular and bodily applications of several drugs that you frequently use: atropine, eserine, pilocarpine, adrenalin and cocaine. Without it, you could not explain why hypothyroids are frequently oversensitive to atropine and pancreatic diabetics to adrenalin; why hyperthyroids frequently have difficulty in adapting themselves to changes of lenses and why they respond badly to orthoptic training for convergence insufficiency; and why pancreatic diabetics often require frequent changes of lenses. Your use of obstetrical pituitrin in herpes zoster, of adrenalin in angioneurotic edema and in glaucoma, and of chondroitin in

migraine are a few examples of endocrinology's contributions. The internal treatment of endocrine disease is generally best not administered by the ophthalmologist. Its knowledge, however, by the ophthalmic consultant as well as the recognition of ocular symptoms which possibly suggest its advisability, may render a marked service to both the general physician and the patient. In the evaluation of many functional ocular symptoms which bring patients to us for relief, the fundamentals of endocrinology are of material aid in the better understanding of our patients as well as their diseases.

Now let us discuss some endocrine therapeutic measures which at the present time apparently have not produced the desired results. The injections of eschatin, which is practically the same as cortin, have been of no value in changing the course of glaucoma in my series, which includes acute, chronic inflammatory and simple cases. Its experimental use as an auxiliary to the accepted myotic or surgical methods of treatment with and without the administration of cevitamic acid should be made by a number of ophthalmologists. I believe that under no circumstances should the proper myotic, surgical, and hygienic treatment be suspended long enough to cause permanent harm while cortin is being experimentally used. In my experience, the use of pituitary and gonadal therapy, including antuitrin S, menformon, etc., in retinitis pigmentosa has not been accompanied by any permanent improvement. Neither has the use of adrenalin seemed to materially change the course of myopia.

Unfortunately, endocrinology easily lends itself to exploitation. The only remedy is its better understanding by more ophthalmologists who will impersonally investigate its chemical, its experimental and its clinical applications, as well as its theoretical and practical uses.

In conclusion, I feel indebted to Dr. Lemoine for the service he has rendered in presenting an interesting subject at an opportune time.

Dr. Louis Bothman, Chicago: In connection with this paper on endocrine disease, I should like very briefly to tell you about a case we first saw at the University of Chicago Clinic in 1934. A boy of eleven years, who was a cretin and had been taking thyroid all his life, came in for routine examination, and it was found that his vision was 20/15 in each eye, and there was no obvious ocular pathology. In 1936 he was brought in because the teacher had noticed his vision was very poor in school. At that time the vision in the right eye was 20/40 and in the left eye even less. The reason for his reduction in vision was obvious.

In both of his eyes the posterior subcapsular area of the lens showed the typical fawn colored granular opacity, which is so frequently associated with hypoparathyroidism, the type of cataract which we see after the parathyroids have been removed at the time of thyroidectomy.

In 1937, about seven months later, he came in again, and at that time he had a bulging right cornea but a normal left cornea. He had further advanced in the cataract in his left eye. The cornea opacity was dense and involved the central three-quarters of the cornea, extending the entire thickness of it and definitely bulging forward as a staphyloma. Ordinarily, with cretinism or hypothyroidism, we do not associate lens opacity or corneal

staphyloma or keratoconus, but in this boy, in addition to his hypothyroidism, there must also have been a hypoparathyroidism, and for that reason I thought it was very interesting.

DR. A. N. LEMOINE, closing: I want to thank Drs. Ruedemann, Bahn, and Bothman for their kind discussion, and I must say I fully agree with Dr. Ruedemann that it is very essential for us to learn to recognize hyperthyroidism early, and frequently the ophthalmologist is the first physician consulted. Many cases of hypothyroidism should be recognized by the ophthalmologist. I thoroughly agree with him in that matter, and I also agree with him that we are not supposed to treat these patients.

This is a plea for a better understanding of the physiology of the endocrine glands in relation to the eye. That is the thing that will differentiate us from the optometrists. Our medical men are referring cases to the optometrists because we are of no value to them as consultants. This is a plea that we remain as doctors, and that we recognize these endocrine disturbances where we can be of assistance as consultants. That is really the main reason for this presentation.

I do not agree with Dr. Ruedemann on the matter that we should just overlook them and let them go. We should qualify ourselves to recognize these conditions, because the eye reveals most of the disturbances more markedly than any other organ in the body, and I should like to make the plea again that we maintain our standing as doctors. Let us not forget that we are physicians and not optometrists, and let us be of some value as consultants.

FURTHER STUDIES ON THE REGENERATION OF THE AQUEOUS IN MAN*

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IF THE aqueous of a human eye is aspirated through a fine hypodermic needle, introduced into the anterior chamber through the periphery of the cornea, several reactive processes are set up within the eye.† The formation of new intraocular fluid and characteristic reactive fluctuations of the intraocular pressure are probably the most important of these processes. The more than 200 anterior chamber punctures which we have performed for diagnostic and therapeutic purposes on the eyes of Chinese patients in the hospital and the out-patient department of the Peiping Union Medical College enable us to make some further contributions to the knowledge of those reactive processes.

THE INTRAOCULAR PRESSURE IMMEDIATELY AFTER THE ANTERIOR CHAMBER PUNCTURE

If the puncture is performed upon an eye with an anterior chamber of average or unusually great depth, the intraocular pressure drops, immediately after the anterior chamber puncture, to a level below the range of the impression tonometers which the clinician uses. The tonometer of Schioetz, for instance, does not register tensions below plus 4 mm. of Hg. Such tonometrical readings are generally recorded as zero. Actually, it is highly probable that a negative intraocular pressure prevails immediately after completely emptying an anterior chamber of average or more than average depth. One of us[1] had the opportunity to determine manometrically this negative pressure in a normal eye that had to be sacrificed because of a malignant orbital tumor and found it to be

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† This procedure is hereafter referred to as anterior chamber puncture (ACP).

minus 5 mm. of Hg. It is a common clinical observation that air or blood from the outside is sucked into the anterior chamber immediately after chamber-opening incisions, a phenomenon which indicates that a negative intraocular pressure exists. The flattening of the cornea, which we have observed and measured ophthalmometrically after anterior chamber punctures, and the contraction of the sclera, which may take place under these conditions, are apparently not sufficient to make up for the acute loss of the fluid contained in anterior chambers of average or greater depth.

A different situation prevails if the puncture is performed upon eyes with shallow anterior chambers. There, despite complete emptying of the chamber, the pressure immediately afterwards may still be positive and of a magnitude which can be measured with a Schioetz tonometer. The amount of fluid removed from these eyes is absolutely — and in all probability also relatively, that is, in relation to the total volume of the eye — smaller than in eyes with average or greater chamber depth. Examples of such eyes with shallow chambers, which are usually glaucomatous or preglaucomatous, are given in Table 1 (taken from P. C. Kronfeld and C. K. Lin, Chinese Medical Journal, 50:1328, 1936).

TABLE 1

Hospital Number of Patient	Volume of Anterior Chamber in cc.	Tension	
		Before Anterior Chamber Puncture	Immediately After Puncture
44603	0.16	28	4
41957	0.125	22	4
53271	0.105	28	7
52284	0.08	22	6
47029	0.06	20	5

This behaviour of the intraocular pressure after anterior chamber puncture in eyes with shallow chambers offers an explanation for the fairly common clinical observation that operative emptying of the chamber of a glaucomatous eye does not bring a very high tension down to normal or to zero. "The eye is still hard," Professor Meller often said after performing an iridectomy for the relief of acute inflammatory glaucoma. The removal of the 0.1 cc. or less of fluid which is contained in the chamber of these cases reduces the total volume of the intraocular contents so little that

the effect upon the tension is bound to be slight. To state that the intraocular pressure immediately after anterior chamber puncture is zero or that it equals the atmospheric pressure is, therefore, inaccurate. The track of a small hypodermic needle through the cornea does not, as a rule, permit the establishment of a pressure equilibrium between the anterior chamber and the outside world. The intraocular pressure immediately after anterior chamber puncture should, therefore, be said to depend upon the relation between the volumes of the anterior chamber and of the entire globe.

The examples given in Table 1 suggest a definite mathematical relationship between the amount of fluid removed and the drop in pressure thus produced. Whether it is possible to apply to the withdrawal of fluid from the human eye the equivalent figures established by Koster,[2] Wessely[3] and others for the injection of fluid into the rabbit's eye will be discussed elsewhere.

THE RESTORATION TIME

Usually the opening in the cornea produced by the puncture needle closes and becomes water-tight immediately. The anterior chamber is slowly restored and the aqueous regenerated; the intraocular tension rises and describes a rather characteristic curve. After a certain time, called the restoration time, the original level of tension is reached. Then the tension rises still further (the phase of relative hypertension) and finally drops again to the original level. The restoration time, the intensity and the duration of the hypertensive phase vary considerably. Some of the factors which account for these variations have been recognized. The restoration time has been shown to depend upon the depth of the anterior chamber prior to the puncture, or, as it were, upon the amount of aqueous which has to be regenerated to restore the status quo ante. These observations were made by one of us on six cases of senile cataract whose ages were from 49 to 65 years.[4] We have had the opportunity to confirm these findings on eyes which were more nearly normal than the aforementioned eyes with senile cataract, namely, on the eyes of 28 cases of either concomitant strabismus or of chronic retrobulbar neuritis.

The eyeballs of these 28 Chinese cases were structurally and functionally normal except for slight degrees of amblyopia in *some* of the patients with strabismus, and for small central scotomata due to optic nerve lesions *with or without* temporal pallor in the cases of retrobulbar neuritis. Especially the eyes in the latter cases may, in our opinion, justly be called nor-

mal ones so far as the vegetative ocular functions are concerned, because in most of them the optic nerve disease had been of very short duration and there was every reason to assume that these eyes had been normal until the outbreak of the optic nerve disease. The behavior of the eyes of the strabismus cases, with or without amblyopia, did not noticeably differ from that of the cases of retrobulbar neuritis. The ages of these patients were from 18 to 32 years.

In these 28 cases we followed the intraocular tension after ACP and found that the restoration time tends to be longer in the cases which had deeper anterior chambers originally. The relation between restoration time and depth of the anterior chamber is not regular enough to allow expression in mathematical terms. For the commoner depths of the chambers, that is, for those whose volume varied from 0.19 to 0.30 cc., the restoration time varies from 90 to 131 minutes and the average of the tension curves is as follows:

T_a	T_0	T_1	T_2	T_3	T_4	T_5
21	0	3	8	18	24	23

These figures signify the tension immediately before (T_a), immediately after the ACP (T_0), and then the tensions every 30 minutes thereafter (T_1, T_2, T_3 and so on).

On 21 eyes of the 28 cases in this group, we performed anterior chamber punctures and, after intervals varying from 45 to 130 minutes, again withdrew and measured the amount of fluid present in the anterior chamber. This amount of regenerated fluid we have tried to correlate with the intraocular tension prevailing at that particular time.

The determination of the volume of chamber fluid cannot be done with great accuracy because it is difficult to aspirate the last trace of the fluid. When, toward the end of the aspiration of the aqueous, the portions of iris which lie next to the needle come in contact with the cornea, traces of fluid may still be present in other parts of the chamber which are no longer in communication with the lumen of the needle. These traces of fluid could be removed by carrying the tip of the needle all around the periphery of the anterior chamber. This would entail considerably greater mechanical irritation of the iris and cornea and would prolong the stay of the needle in the anterior chamber. Both these factors would introduce new possibilities of error. We have, in this series, stopped aspirat-

ing when the iris next to the tip of the needle touched the cornea. The amount of aqueous left in the anterior chamber probably did not exceed 0.01 cc.

TABLE 2

O.P.D. or Hospital Number	Tension in mm. Hg.		Volume of Anterior Chamber in cc.		Interval Between the 2 Punctures
	Before the 1st Puncture	Before the 2nd Puncture	At the 1st Aspiration	At the 2nd Aspiration	
56738	21	19	0.25	0.25	95
58931	17	19	0.29	0.28	126
57520	22	20	0.28	0.28	106
58124	19	20	0.23	0.235	109
58580 o.s.	13	13	0.265	0.255	126
376695	19	21	0.22	0.225	95
368376	20	19	0.30	0.31	130
370580	14	15	0.275	0.28	127

TABLE 3

O.P.D. or Hospital Number	I Original Volume of the Anterior Chamber in cc.	II Amount of Fluid Present in the Anterior Chamber 1 Hour After the 1st Puncture, in cc.	$\frac{II}{I} \times 100$
357690	0.180	0.190	106
364304	0.210	0.210	100
57957	0.210	0.197	94
56678	0.220	0.210	96
57520	0.240	0.230	96
57957	0.243	0.195	80
55620	0.246	0.208	85
56738	0.255	0.200	78
364304	0.258	0.188	73
57520	0.260	0.230	80
56738	0.270	0.198	73
368656	0.300	0.172	57
		average 0.202	

In the cases listed in Table 2, we performed the second anterior chamber puncture at a time when the intraocular pressure had returned approximately to the original level. The data thus obtained

show that at the time at which the tension is restored to its original level, the anterior chamber is also restored to its original depth, or, in other words, that the restoration time for the aqueous is approximately the same or perhaps shorter than the restoration time for the intraocular tension. The data contained in Table 3, which gives the amounts of aqueous present in the anterior chamber one hour after the puncture, show independence of the rate of fluid regeneration from the original chamber volume. According to these data, the regeneration of the aqueous takes place at the same absolute rate in all the eyes represented in the table, the result being that an originally deeper chamber is restored after a longer interval than an originally shallower chamber. The volumetric findings thus confirm the results of the tonometric studies. Throughout these studies a striking parallelism between intraocular pressure and anterior chamber volume has been encountered. We have not run across any marked inconsistencies in the sense that, for instance, the chamber volume was completely restored, whereas the intraocular tension was still considerably below the original level. An explanation for such inconsistencies, if they did occur, would be the assumption first made by Hagen that intraocular fluid is given off by the vitreous and poured into the anterior chamber. The possibility that such a process takes place during the earlier phases of the regeneration of the aqueous cannot be denied. The actual demonstration of such inconsistencies is difficult because of the inaccuracy of the tonometric as well as the volumetric methods used.

THE HYPERTENSIVE PHASE

The finer details in the mechanism of the hypertensive phase are not known. The factors which probably play a part are: reactive alterations of the intraocular vascular pressures, of the vessel caliber and of the vascular permeability (chiefly in the anterior uvea) with secondary effects upon the rate of formation, quality and absorption of the intraocular fluid. It may be assumed that this whole complex of reactive changes is most pronounced *shortly* after the puncture. The hypertensive phase which the tonometer registers two hours after ACP would then not express the entire extent of those reactions, but only their "tail-end." The rise of tension during the restoration time would be due to the reformation of aqueous as well as to other reactive factors. If these assumptions are correct, eyes with short restoration times should

show more pronounced hypertensive phases than eyes with long restoration times.

The tonometric studies on the 28 cases of this series did not bring out such a relationship. The hypertensive phase expressed in rise of tension above the original level appears to be independent of the length of the restoration time and, therefore, also independent of the original chamber volume. To explain this independence of the hypertensive phase from the original chamber volume, one might think of a safety valve action of the canal of Schlemm which would tend to curtail the hypertensive phase and to make it uniform. Another possibility would be that the entire amount of the reactive alterations is proportional to the amount of fluid withdrawn, but that, in the cases of deeper chambers, because of the longer restoration time, a later and, therefore, less pronounced phase of the reactive alterations would cause the reactive hypertension. To put these considerations to the test, we have compared the tension curves after partial and after total emptying of the anterior chamber on the same patient. On the two eyes of a Chinese male patient (P.U.M.C. Hospital, No. 45842, age 31), we performed five anterior chamber punctures (see Table 4). The patient's eyes were normal except for the presence of small central

TABLE 4

Date of ACP	Eye	Amount of Aqueous Withdrawn in cc.	Intraocular Tension in mm. Hg.							Highest Tension $-T_a$
			T_a	T_0	T_1	T_2	T_3	T_4	T_5	
9/1/34	R.E.	0.27	23	0	0	6	21	33	29	10
9/25/34	R.E.	0.12	20	5	12	18	26	27	27	7
9/4/34	L.E.	0.27	20	0	0	6	19	27	30	10
9/19/34	L.E.	0.13	25	6	13	20	27	27	27	2
10/4/34	L.E.	0.16	18	0	6	14	24	25	22	6

scotomata due to lesions in the retrobulbar portion of the optic nerves (healed retrobulbar neuritis). The patient's vision was 6/60 in each eye. The data contained in Table 4 show that the hypertensive phase as expressed in rise of tension above the original level was less pronounced after the partial than after the complete emptyings, despite the shorter restoration time in the former in-

stance. The conclusion seems justifiable that complete emptying of the chamber is a stronger stimulus than partial emptying, as far as the reactive alterations are concerned.

SUMMARY

The intraocular tension of human eyes immediately after anterior chamber puncture depends upon the relation between the volume of the anterior chamber and the volume of the entire globe.

The time interval necessary for restoration of the intraocular pressure to the original level depends, in nearly normal human eyes, upon the original volume of the anterior chamber.

The rate at which, in nearly normal human eyes, new intraocular fluid is formed after anterior chamber puncture appears to be independent of the original chamber volume.

The intensity of the reactive ocular changes which follow anterior chamber puncture and manifest themselves as the hypertensive phase seems to be proportional to the intensity of the eliciting stimulus, that is, to the amount of fluid withdrawn.

REFERENCES

1. Kronfeld, P. C.: Arch. of Ophth. 1:457, 1929.
2. Koster, H.: Arch. f. Ophth. 41:153, 1895.
3. Wessely, K.: Arch. f. Augenh. 60:8, 1908.
4. Kronfeld, P. C.: Arch. of Ophth. 9:805, 1933.

DISCUSSION

DR. DERRICK VAIL, Cincinnati: It is with great hesitancy that I stand before you to discuss a paper of this kind, and yet as the paper went on it seemed to me there were certain clinical attributes which might be explained or clinical findings which might be explained by Dr. Kronfeld's contribution.

As I mentioned in discussing Dr. Davis' paper in these cases of hyphema in the chamber following cataract extraction, I found intraocular tension to be elevated on the third or fourth day in a certain number of cases, and it seems to me that the reason the intraocular tension has been elevated at that point has been a rather sudden, or at least a maximum increase of the amount of vitreous into the anterior chamber due to the sudden closure of the wound.

I should like to ask Dr. Kronfeld's reaction to that idea.

One other point along this same line is that some of these patients who show a high fever on the fourth or fifth day also show a deep chamber on the third day, or, in other words, the high fever followed a day on which the anterior chamber was usually deep, deeper than it had been prior to operation. This is also thought-provoking, and perhaps Dr. Kronfeld's investigations, or at least present or future ones, might throw some light on this point.

Dr. Peter C. Kronfeld, closing: I am glad that Dr. Vail could offhand think of clinical applications of my very humble academic studies. An unusually pronounced hypertensive phase after anterior chamber puncture is, as far as I know, characteristic of a glaucomatous eye. Conditions in the cases mentioned by Dr. Vail are, however, very complex, and I must say that I can offer no explanation.

Section on Otolaryngology

The American Academy
of Ophthalmology and Otolaryngology
1 1 1
Forty-Second Annual Meeting
Chicago 1 1937

AN EXPERIMENTAL STUDY OF THE RELATION OF IRRITATION IN THE PARANASAL SINUS REGION TO CERTAIN VASOMOTOR CHANGES*

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THE PROBLEM of pain in the mastoid region, neck, base of the head and contiguous areas from infection in the region of the sphenopalatine ganglion has received much attention. There is no need to review the literature at the present time. Various explanations of this syndrome, called after Sluder who first pointed it out clearly, have been offered by a number of students of the problem. In 1936, two of the present authors,[1] on the basis of anatomical and experimental considerations, offered the hypothesis that Sluder's syndrome is due to vasomotor changes in the areas of pain brought about by reflexes induced through irritation by sinus disease of the nerves in the region of the sphenopalatine ganglion. This hypothesis assumed that reflex ischæmia is produced, giving rise to local pain impulses. These are presumably transmitted to the central nervous system by the segmental cutaneous nerves of the region involved. The hypothesis was strengthened by the fact that human patients with typical symptoms were relieved of their pain by injection of novocain into the skin of the mastoid region. The nervous pathways thought to be involved are the nasal and palatine branches of the sphenopalatine ganglion, the great superficial petrosal nerve, connections within the medulla oblongata with the reticulo-spinal tract and, through the latter, connections with the segmental autonomic nerves in the lower cervical segments of the cord which supply the vasomotor innervation of the arteries of the regions involved in Sluder's syndrome.

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It appeared feasible to test this hypothesis further by experimental methods in animals. While there is no adequate method of determining whether, in animals, pain is produced in the regions under consideration the vasomotor effects could at least be tested.

Thanks to the continued financial support of the Research Committee of the Council of the American Academy of Ophthalmology and Otolaryngology, this investigation has been facilitated.

MATERIAL AND METHODS

Nasal fibers from the maxillary nerve may also be involved in this reflex arc. Allen[2] has shown experimentally that faradic stimulation of such fibers produces vasoconstriction and increased blood pressure. To simplify the present experiment as much as possible the maxillary nerve was sectioned proximal to its connections with the sphenopalatine ganglion in all cases before stimulation of the sphenopalatine region with a faradic current was undertaken.

The further procedure consisted in determining the vasomotor effects of such stimulation after various links in the supposed nervous pathway above named had been sectioned. To this end the spinal cord was sectioned between the 6th and 7th cervical segments in some of the animals and the cervical sympathetic trunk was sectioned in others. After each such operative procedure the sphenopalatine ganglion region was stimulated by faradic current and the vasomotor effects in the ears of rabbits were noted and photographed. The ears were used because the blood vessels can easily be seen and photographed by transmitted light, especially after the hair is shaved off. The chief blood supply to the ear is from a branch of the posterior auricular artery, which also supplies the mastoid region, so that vasomotor effects involving the one vessel should also affect the other, to some extent at least. This region, in the human, receives its cutaneous innervation chiefly from the 2nd and 3rd cervical nerves, with the cutaneous branch of the facialis supplying a small area. The vasomotor innervation of the region is presumably by fibers from the superior cervical ganglion reaching the vessels named through the 2nd and 3rd cervical nerves. There is every reason to believe that this innervation, both cutaneous and vasomotor, is essentially the same in the rabbit, although it has not been worked out with the same anatomical detail as in the human.

Albino rabbits three to five months old, weighing between 1.8 and 2.5 kilograms were used in the experiments. All operations were carried out under nembutal anesthesia with as near aseptic conditions as was possible in the laboratory.

The operations were done in successive stages. The technique of the first operation was as follows: After preparation of the operative field an incision was made through the skin along the inferior border of the zygomatic processes of the zygomatic and the maxillary bones. Attachments of orbital fascia along the upper border and the masseter muscle along the lower border of the bone were dissected away. The zygomatic bone was removed by bone forceps. The fibers of the pterygoid muscles were separated by blunt dissection, and the maxillary division of the fifth cranial nerve exposed. The internal maxillary artery passing very close to the nerve was separated and the nerve sectioned as far posteriorly along the wall of the orbit as possible, so as to leave intact the sphenopalatine ganglion and its connections. The Vidian nerve and the posterior nasal nerve lying deeper than the maxillary nerve along the floor of the orbit was not disturbed in this operation. Two to five days were allowed for recovery after the above operation before proceeding with the remainder of the experiments.

An attempt was made at first to study the vessels in the ear of the operated side with the use of the transparent window devised and described by Clark et al.[4] This was abandoned in favor of photographing the entire vascular field with a magnification of about 2.77 to 3.7 times and observing the "gross" vascular changes from the photographic negatives and prints. The photographs were taken of the shaved ear on the operated side before and after stimulation of the nasal mucosa on the same side, without changing the photographic apparatus in any way. The photographs were in serial order to include those before and after stimulation of the nasal mucosa after each operative stage.

The complete operative stages were as follows: Section of the maxillary division of the 5th cranial nerve with an allowance of 2-5 days for recovery of the animal; followed by hemisection of the cervical spinal cord between the sixth and seventh segments. Complete section of the cervical spinal cord at the same level; and in some of the animals (see table 1) complete section of the cervical sympathetic trunk between the middle and inferior cervical ganglia.

TABLE I — *Results of stimulation of nasal mucosa on the operated side of the animal as recorded by photographs of the vascularity of the ear on the same side.*

Animal No.	Sex	Wt. in Kilos	Operation Section	Time from previous operation	Photographic results: Observations of negatives of vascular fields after stimulation of nasal mucosa on operated side
1	M	2.16	Maxillary Nerve	died	
2	M	2.22	Maxillary Nerve	3 days	constriction of vessels
3	M	2.22	Maxillary Nerve	5 days	constriction of blood vessels
			Sympathetic Trunk	1 hr.	dilation of vessels remained so after stimulation
			Sympathetic Trunk	4 days	no change in vascular area
			Complete cord	6 days	no change in vascular field
4	M	2.5	Maxillary Nerve	5 days	constriction
			Hemisection sp. cord	1 hr.	no change
			Complete cord	1 hr.	no change
5	M	2.5	Maxillary Nerve	5 days	constriction
			Hemisection cord	1 hr.	no change
6	F	2.5	Maxillary Nerve	3 days	constriction
			Hemisection cord	1 hr.	slight constriction noted
			Complete cord	1 hr.	slight constriction noted
			Cerv. symp. Trunk	3 hrs.	no change
7	M	1.81	Maxillary Nerve	2 days	constriction
			Hemisection cord	1 hr.	no constriction
			Complete cord	2 hrs.	no constriction
			Cerv. symp. Trunk	4 hrs.	no constriction

No. 6 — Section of the cervical spinal cord below the level of the seventh segmental vertebra below the level of the second thoracic vertebra.

The photographs taken of the vascular field of the ear before and after stimulation of the nasal mucosa on the animal after the maxillary nerve section were used as a standard to compare the photographs taken after the subsequent operations. A time interval of approximately one hour was given after the operations before the stimulation and photographing.

Stimulation of the nasal mucosa was done with the use of a copper electrode, the current being derived from two dry cells led in turn into a reduction coil to produce a continuous faradic cur-

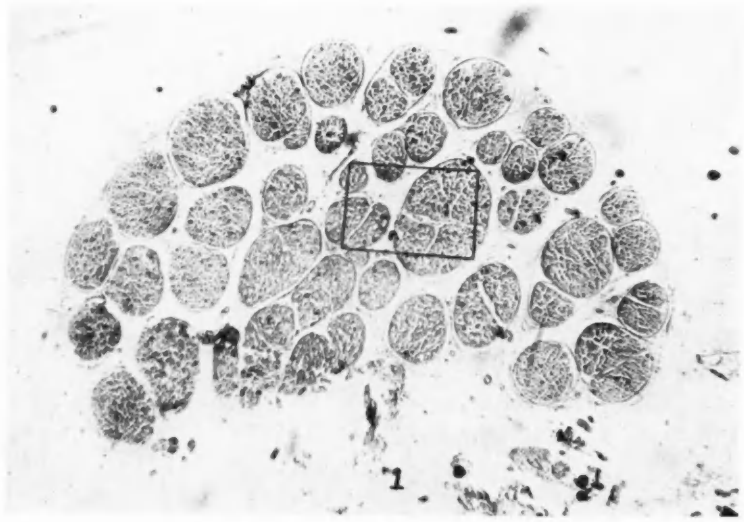

FIG. 1 — (Larsell-Barnes-Fenton).

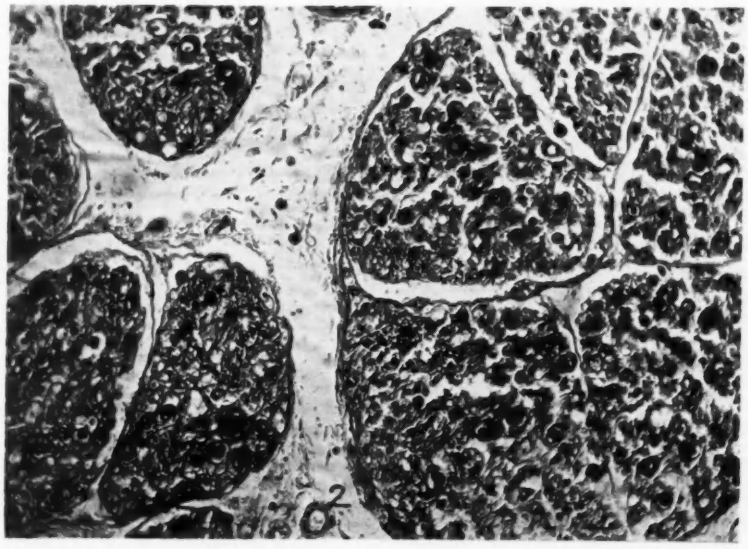

FIG. 2 — (Larsell-Barnes-Fenton).

FIG. 3 — (Larsell-Barnes-Fenton).

FIG. 4 — (Larsell-Barnes-Fenton).

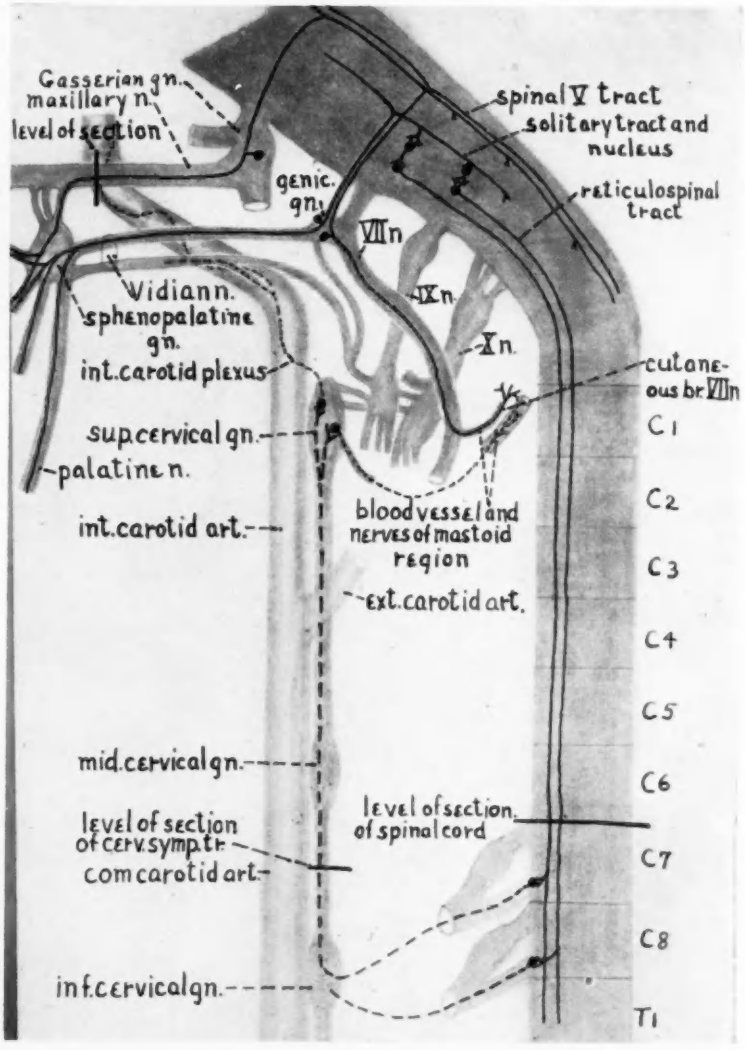

FIG. 5 — (Larsell-Barnes-Fenton).

rent. A medium strength current was used in most instances, but a strong current was substituted after section of the cervical spinal cord, and of the cervical sympathetic trunk.

An autopsy was performed on each animal after each series of experiments verifying the operative technique. A portion of the peripheral end of the sectioned maxillary division was removed and subsequently stained with osmic acid to observe the degeneration of the nerve fibers (figs. 1 and 2).

RESULTS

Reference to Table I will show that quite uniform results were obtained in animals 2, 3, 4, 5, and 7. Animals 1 and 8 were not utilized because of faulty technique in operation and because of difficulties with anæsthesia.

In all animals, uniform results were obtained after section of the maxillary division of the 5th cranial nerve, namely, constriction of the vessels after stimulation of the nasal mucosa on the operated side (figs. 3 and 4). This indicates there are other afferent fibers involved in the innervation of the mucosa of the nose besides those carried by the fifth cranial maxillary division, thus causing vasoconstriction. These are carried in the posterior nasal, great superficial petrosal and possibly the deep petrosal nerves, as suggested by Larsell and Fenton.[1]

The vasomotor response was abolished by section of the cervical spinal cord at the level of the sixth spinal segment, or by section of the cervical sympathetic trunk between the middle and inferior cervical sympathetic ganglia. Only in one animal was there a failure to abolish the vasoconstriction after section of the cervical spinal cord. Subsequent autopsy showed that in this animal the section had been made at a level where it would fail to completely interrupt the outflow to the cervical sympathetic trunk in that region, i.e., between the 7th and 8th cervical segments.

The afferent fibers in the nasal region coursing in the seventh nerve and ending in the solitary tract and its nucleus undoubtedly make synaptic connection in the reticulo-spinal tract. The impulses passing along this tract make connections with the preganglionic fibers of the sympathetic vasoconstrictors which arise from the lower cervical and upper thoracic portion of the spinal cord, thus affecting the arteries of the ear and adjacent structures.

It was possible to eliminate the impulses carried by the reticulospinal tract by section of the cervical spinal cord above the sym-

pathetic outflow. The result after section was complete lack of vasomotor response when the nasal mucosa was stimulated on the operated side. Further, after section of the cervical sympathetic trunk, with the spinal cord intact, vasomotor responses failed to appear. Section of the spinal cord below the level of the autonomic outflow to the head region failed to affect vasomotor responses in the ear, but they were abolished in the same animal after section of the cervical sympathetic trunk.

TABLE II — *Percentage variation in size (diameter) of the blood vessels of the ear after stimulation of the nasal mucosa on the same side.*

Animal	Nerve Section	Percentage of Variation
2	Maxillary	20-25 constriction
3	Maxillary	14-27 constriction
3	Sympathetic	34 dilatation
4	Maxillary	25 constriction
5	Maxillary	35 constriction
6	Maxillary	17 constriction
7	Maxillary	12 constriction

The degree of vasomotor change under the various conditions of the experiment is indicated in Table II, compiled from data obtained by measuring the same blood vessel in the photographs taken before and after stimulation. The vessels were measured at the same point in their course. The variation is expressed in percentage of diameter.

BIBLIOGRAPHY

1. Larsell, O., and Fenton, Ralph A.: *Sympathetic Innervation of the Nose.* Archives of Otolaryngology, 24:687-695, Dec. 1936.
2. Allen, W. F.: (a) *Formatio Reticularis and Reticulospinal Tracts: their visceral functions and possible relationships to tonicity and clonic contractions,* J. Washington Acad. Sc., 2:492, 1932;
(b) *Effect on Respiration, Blood Pressure, and carotid pulse of various inhaled and insufflated vapors when stimulating one cranial nerve and various combinations of cranial nerves: I. Branches of the trigeminus affected by these stimulants,* Am. J. Physiol., 87:319, Dec. 1928.
3. Fenton, R. A., and Larsell, O.: *The Mechanism of Pain Transmission in Certain Types of Otalgia.* Annals of Otology, Rhinology and Laryngology, vol. 37, 1928.
4. Clark, E. R.; Smith, K.; Rex, H. T.; Williams, R. O.: Proc. Soc. Exp. Biol. & Med., 26:745-747; Anat. Rec., Vol. 28, No. 4:281; Am. J. Anat., Vol. 41, No. 3:447; Anat. Rec., Vol. 40, No. 1:41.

AN EXPERIMENTAL STUDY OF STREPTOCOCCUS INFECTION OF THE LUNGS FROM THE PARANASAL SINUSES*

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THE RELATION of certain types of pulmonary infection to drainage from the paranasal sinuses has been emphasized by Mullin[1] in a series of papers. The various possible routes by which such drainage may reach the lungs have been discussed by Mullin,[1] Lawson[2] and others. More recently, the results of experimental studies[3] have shown clearly that probably the most important pathway is a combined lymphatic and blood vascular route from the sinus membranes to the pulmonary capillary bed via the right heart. These conclusions were reached as a result of injecting trypan blue into the sinuses and related regions under conditions which precluded the tracheal pathway to the lungs. Later, at various intervals, the trypan blue was found in phagocytes in the lungs at various points along the route indicated.

On the basis of these results, experiments were undertaken to determine if cultures of streptococcus, introduced into the paranasal sinuses and the lymph nodes receiving drainage from these sinuses, follow the same route in animals. The present paper deals with these experiments.

MATERIAL AND METHODS

Three series of animals, including cats and rabbits, were inoculated with cultures of streptococcus. As a rule, inoculation was made into the frontal sinuses or into the paralaryngeal lymph nodes which drain these sinuses. The maxillary sinuses were inoc-

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ulated in two rabbits. The animals were killed after periods of 1 to 144 hours. The paralaryngeal lymph nodes, lungs, parts of liver and of spleen were removed with aseptic precautions and pieces were taken from each for bacteriological culture. In some cases mediastinal lymph nodes also were removed. Other pieces of the various organs were fixed in formol-Zenker solution for histological study, after staining by methods calculated to reveal the bacteria in the tissues. For this purpose we found the MacCallum modification of Goodpasture's basic fuchsin and crystal violet stain the most useful. Bacteriological cultures of blood from the right heart were routinely made.

The lungs of most of the animals showed more or less evidence of infection at autopsy, ranging from small spots to considerable consolidation. These gross signs were not regarded as necessarily due to the experimental inoculations since cats and rabbits frequently show pulmonary lesions without inoculation. Only when the β-streptococcus was recovered by bacteriological methods or morphologically similar forms were seen in stained sections was the result regarded as positive. There is no attempt to indicate in Table I the relative number of organisms, although this varied in different animals. The results are regarded as entirely qualitative and as demonstrating anatomical pathways rather than the degree of infection or the reactions of the tissues. The histological procedure served to determine whether the organisms shown by bacteriological culture methods were present in the bronchi or in the parenchyma of the lungs. They were virtually absent from the bronchial passages, but were found in the parenchyma and the lymphatics, as more fully described below. The tracheobronchial route of entry cannot be as rigidly excluded for bacteria as for trypan blue in the earlier experiments, because the insertion of a tracheal cannula to eliminate material from the upper respiratory region in itself favors bacterial contamination of the air passages. Cannulæ, therefore, were not used.

In spite of precautions to prevent dripping of inoculated material from the frontal sinuses, especially into the nasal cavity and thence into the trachea, this possible pathway to the lungs must be considered. The precautions consisted of injury to the sinus mucosa in the region of the ostia in a manner designed to produce swelling of the membranes and thus closure of the ostia. The position of the sinus openings into the nasal cavity and the posture of the head in the animals used also are such as to prevent dripping

into the nasopharynx or trachea. It is believed on these grounds as well as on the ground of histological study of the bronchi and lungs that the tracheal route was negligible in these experiments. In the animals inoculated directly into the paralaryngeal lymph nodes the possibility of the tracheal route was entirely eliminated.

The first group of animals, designated Series A, consisted of five cats. These were inoculated with a saline suspension from 24 hour blood agar culture of a feebly hemolytic β-streptococcus isolated from the frontal sinus of a human patient. Approximately one-half million to one million organisms were introduced into each of the animals of this series by the routes indicated in Table I, and were allowed to act for the time periods also indicated in the table.

It was soon apparent that the paralaryngeal lymph nodes filtered the bacteria pretty thoroughly when inoculated into the region of the paranasal sinuses. In Series A, therefore, which was inoculated with relatively small doses, the results by bacteriological methods for the organs cultured were negative, as indicated in Table I.

In Series 37-C, consisting of 12 cats, and in Series 37-R, consisting of 6 rabbits, a new culture of a β-streptococcus was used. The concentration was made greater than in the culture used in Series A by suspending the sediment from 25 cc. of Hartley broth culture of 24 hours' duration in 1 cc. of saline and injecting this mass into sinuses or lymph nodes. The procedure gave individual dosages of approximately 40 million to 6 billion organisms, as shown in Table I. The Hartley broth cultures were made as for Series A, quantitative blood cultures being made in blood agar plates. Pieces of lymph nodes, lung, liver and spleen were also fixed for histological study. Nembutal anæsthesia was used during all the operative procedures, thus avoiding any effect of ether on the respiratory tract.

OBSERVATIONS

Hartley broth cultures of lung, liver, spleen, heart blood and mediastinal lymph nodes were negative in Series A, as already indicated. Histologically two of the animals showed streptococci in phagocytes within the paralaryngeal lymph nodes and two others showed streptococci in the lungs. In Series 37-C and 37-R, which received massive doses of the organism, the paralaryngeal lymph nodes were apparently unable to cope with the invasion and many

TABLE 1 — *Experiments on Inoculation of Streptococcus into Paranasal Sinuses and Paralaryngeal Lymph Nodes, with Effects on Various Other Organs.*

Animal Number	Route of Inoculation	Approx. No. of Streptococci	Inoc. Hours Before Death of Animal	RESULT OF CULTURE						HISTOLOGICAL				REMARKS
				Paralaryngeal Lymph Node	Lung	Liver	Spleen	Heart Blood	Mediastinal Lymph Node	Paralaryngeal Lymph Node	Lung	Liver	Spleen	
CATS														
A-2	Post. Wall Phar.	0.5 Mil.	Ca. 3	N.C.	—	—	—	—	—	+	Con.	Con.	Con. +	Found dead in cage.
A-4	Frontal Sinus	1 Mil.	Ca. 4	N.C.	—	—	—	—	—					Found dead in cage. No histological study.
A-3	Frontal Sinus	1 Mil.	96	N.C.	—	—	—	—	—	+	—			Lymph nodes enlarged. Lymph nodules in lung enlarged.
A-1	Post. Wall Phar.	0.5 Mil.	120	N.C.	—	—	—	—	—		+			Lymph nodes enlarged.
A-5	Frontal Sinus	1 Mil.	144	N.C.	—	—	—	—	—		+			Lower lobes of lungs consolidated. Lymph nodes, spleen, liver enlarged.
37-C-6	Region of L. Par. L. N.	1 Bil.	1	—	—	—	—	—	—		+		+	Killed 1 hr. after inoculation.
37-C-2	R. and L. Par. L. N.	2 Bil.	Ca. 3½	N.C.	—	—	—	—	—		+	+		Found dead in cage 3½ hrs. after inoculation.
37-C-8	R. and L. Par. L. N.	1 Bil.	3	N.C.	—	—	—	—	N.C.	+	+	+	+	Lung showed large mottled areas.
37-C-5	R. and L. Jar. L. N.	2 Bil.	5	+	+	+	+	$\frac{2}{P}$ CC.	—	+	+	+	+	
37-C-7	Region of Par. L. N.	2 Bil.	5	N.C.	+	+	+	$\frac{1200}{P}$ CC.	—	+	+	+	?	Lungs mottled.
37-C-1	R. and L. Par. L. N.	2 Bil.	24	+	+	+	+	+	+	+	+		+	Found dead in cage. Regional lymph nodes greatly enlarged. Lungs consolidated.
37-C-4	L. Par. L. N.	0.4 Bil	48	+	+	—	—	—	+	+	+			Regional lymph nodes enlarged. Spleen dark. Lungs normal grossly.

Animal Number	Route of Inoculation	Approx. No. of Streptococci	Inoc. Hours Before Death of Animal	RESULT OF CULTURE						HISTOLOGICAL				REMARKS
				Parapharyngeal Lymph Node	Lung	Liver	Spleen	Heart Blood	Mediastinal Lymph Node	Parapharyngeal Lymph Node	Lung	Liver	Spleen	
37-C-3	L. Par. L. N.	40 Mil.	72	+	+	+	+	—	—	+	+	+	+	Regional lymph nodes enlarged. Spleen dark. Lungs normal grossly.
37-C-10	L. Fr. Sinus	1.5 Bil.	2½	—	+	—	—	—		+	?		+	Died 2½ hrs. after inoculation. Spleen greatly enlarged.
37-C-11	L. Fr. Sinus	6 Bil.	Ca. 20		—		—	—	N.C.					No gross pathology. Apparently died from anæsthetic.
37-C-9	L. Fr. Sinus	6 Bil.	48	—				—	—	+	+	+	+	Spleen large and dark. Regional lymph nodes large.
37-C-12	L. Fr. Sinus	4 Bil.												Died from anæsthetic.
RABBITS														
37-R-4	R. and L. Par. L. N.	2 Bil.	1	+	—	—	—	—	N.C.	+	+	?		Died 1 hr. after inoculation (anæsthetic). Regional lymph nodes filled with fluid.
37-R-1	Region of R. and L. Par. L. N.	2 Bil.	2½	N.C.	—	—	N.C.	—	N.C.		+			Died Ca. 2 hrs. after inoculation. Lymph nodules in lung enlarged.
37-R-3	Region of R. and L. Par. L. N.	2 Bil.	3	N.C.	Con.	+	+	$\frac{400}{+}$ P CC.		+	+	+	+	Petechial spots in lung.
37-R-2	Region of R. and L. Par. L. N.	2 Bil.	7	N.C.	+	+	+	$\frac{4}{+}$ P CC.	N.C.	+	+	+	+	
37-R-6	R. Max. Sinus	3 Bil.	24	—	+	+	+	—		+	+			Regional lymph nodes enlarged.
37-R-5	R. Max. Sinus	3 Bil.	48	Con.	Con.	—	—	—						Paralaryngeal nodes enlarged. Lungs mottled at margins.

KEY: Con. = Contaminated. N.C. = Not Cultured.

of the organisms filtered through them into the efferent lymphatics and finally into the blood stream. In these animals the lungs, liver and spleen gave positive cultures as early as 5 hours (in one rabbit, 3 hours) after inoculation. Most of them, as indicated in the chart, showed streptococcus histologically even when the results of culture methods were negative. This raises the question of viability of the streptococcus after ingestion by phagocytes. Possibly the ingested organisms shown in stained sections were not viable, but we have no evidence save that the organs in which they were found did not give positive results by bacteriological methods. Another possibility must be recognized, namely, that the pieces of the various organs cultured may not have been reached by the organisms. In either case it is clear, as would be expected, that large numbers of bacteria inoculated into either sinuses or paralaryngeal lymph nodes are more effective in causing viable bacteria to reach the lungs and other organs than are small inoculations. This suggests that positive results, when obtained by bacteriological methods, may have been due to the presence of free streptococci within the organs cultured. That such are present in the lung parenchyma of some of the animals is shown in figure 1, both as individual organisms and in small masses, some of which suggested small thrombi, apparently in the capillaries.

Streptococcus-laden phagocytes were also found in the lymph nodules along the bronchial walls within the lungs, in bronchial lymphatics (figure 2), and, in small numbers, in the lymphatics of the pleura. They thus appear in the same anatomical locations within the lung as did the trypan blue laden phagocytes of the earlier experiments. The bronchial lymph nodes, usually small in the cat, were enlarged, especially in the animals kept alive for some time after inoculation. There was also an increase of the normally relatively large bronchial lymph nodules of the rabbit's lung.

Sections of paralaryngeal lymph nodes, liver and spleen fixed between 1 and 24 hours after inoculation, also show free streptococci in the tissues (fig. 3) as well as ingested bacteria (figs. 4 and 5). The number varies with the organ and animal, as well as with the time period after inoculation.

The series of animals is too small to permit fixed conclusions regarding the time factor in clearing the blood stream of the inoculated bacteria. The results clearly show, however, that the blood stream is an important link in the dissemination of the organisms from the sinus region to the lungs, liver, spleen, and presumably

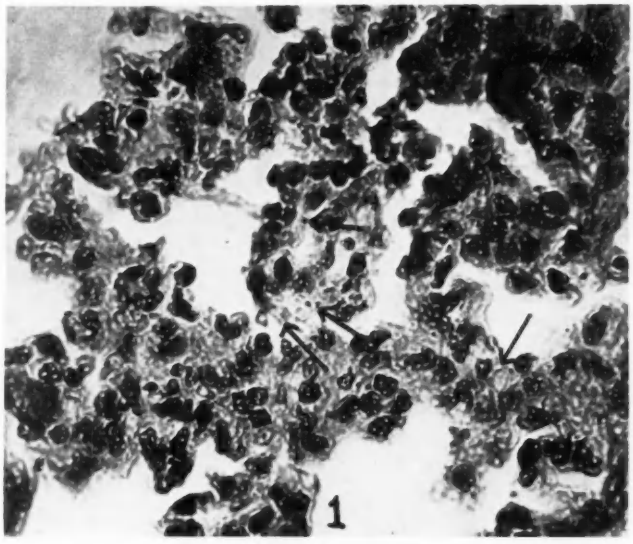

FIG. 1 — (Larsell-Veazie-Fenton) — Streptococci of lung of cat in which frontal sinus had been inoculated with a dosage of concentrated culture.

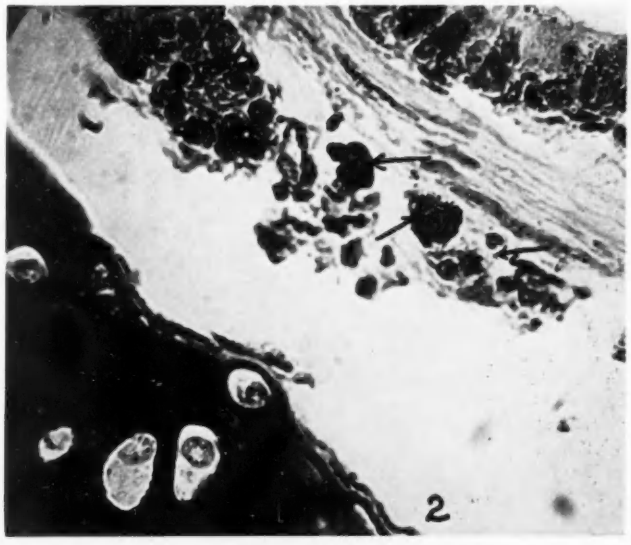

FIG. 2 — (Larsell-Veazie-Fenton) — Phagocytes laden with streptococci in bronchial wall of same animal.

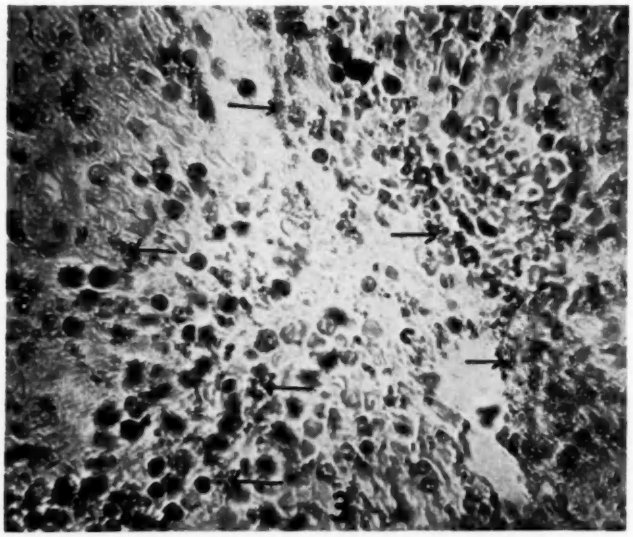

FIG. 3 — (Larsell-Veazie-Fenton) — Paralaryngeal lymph node of rabbit with masses of free streptococci in lymph node sinuses 3 hours after inoculation into the node.

FIG. 4 — (Larsell-Veazie-Fenton) — Streptococci-laden phagocytes in peripheral sinus of paralaryngeal lymph node after inoculation of concentrated culture into the frontal paranasal sinus of cat.

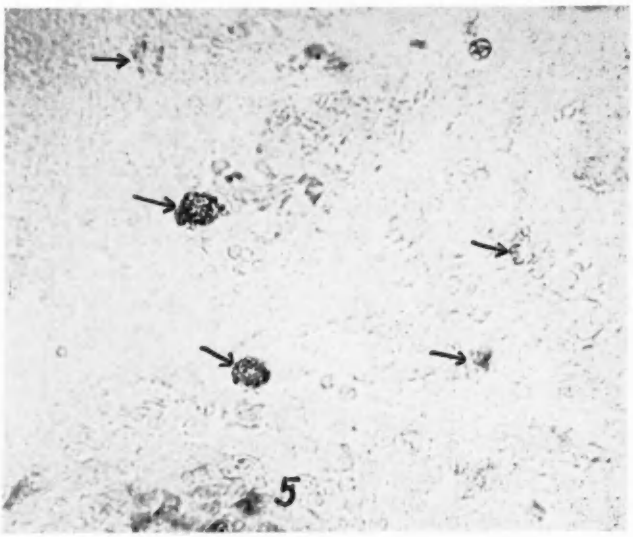

FIG. 5 — (Larsell-Veazie-Fenton) — Streptococci-laden phagocytes in deep sinuses of paralaryngeal lymph node of cat after inoculation into the lymph node.

to other parts of the body which have not been examined in the present study. Under the conditions of the experiment the most obvious route from the sinus membranes is that via lymphatics to paralaryngeal lymph nodes, thence through the paratracheal lymph vessels to the great veins and on into the right heart, from which blood was taken for culture.

It would be of interest to compare cultures of blood from the right heart with similar cultures from the left heart with reference to number of organisms, etc., but this has not been done. The results both by bacteriological methods and by histological study of the liver and spleen indicate that the lungs do not constitute so effective a filter for streptococcus as they do for trypan blue, since relatively little of the latter substance could be found in the liver and the spleen in the earlier experiments. On the basis of blood cultures, it appears that the blood stream is pretty well cleared of organisms, under the conditions of these experiments, in about 24 hours, since only one animal of the 23 used showed positive blood culture as late as 24 hours after inoculation.

SUMMARY AND CONCLUSIONS

Viable streptococcus, as shown by bacteriological methods, and checked as to the pathway followed by histological control, can reach the lungs from the paranasal sinuses and the related regional lymph nodes.

The number of organisms reaching the lung is much less when the sinuses are inoculated than when the bacteria are introduced directly into the paralaryngeal lymph nodes, which normally serve as the filters for the lymph stream from the sinus region.

The fact that streptococci reach lungs, liver and spleen from the paralaryngeal lymph nodes, either when sinuses or lymph nodes are inoculated, indicates that the anatomical path must be via the paratracheal lymph vessels to the great veins, thence into the right heart, then into the pulmonary capillary bud. From this point they may reach the pulmonary tissues or may be carried by the blood stream to other parts of the body. Most of the organisms that pass through the lungs are apparently filtered out of the blood stream by the spleen and by the Kupffer cells of the liver.

The thanks of the authors are due to the research committee of the Council of the American Academy of Ophthalmology and Otolaryngology for its financial support of this research.

BIBLIOGRAPHY

1. Mullin, W. V.: *The Lymph Drainage of the Accessory Nasal Sinuses,* Tr. Am. Laryng., Rhin. & Otol. Soc. 25:73, 1919;
The Relation of Sinus Disease to Diseases of the Chest, Tr. Mid-West. Sect. Am. Laryng. Rhin. & Otol. Soc. 26:488, 1920;
The Relation of Sinus Infection to Disease of the Lower Respiratory Tract, J.A.M.A., 87:739, Sept. 4, 1926;
A Review of Sinus Chest Infections, Ann. Otol., Rhin. & Laryng., 41: 794, 1932.
2. Lawson, L. J.: *The Role of Nasal Accessory Sinus Membranes in Systemic Infection and Toxemias,* Ann. Otol., Rhin. & Laryng., 39:159, 1930.
3. Larsell, Olof, and Fenton, Ralph A.: *Lymphatic Pathways from the Nose,* Arch. Otolaryng., 24:696, 1936.

MALIGNANT DISEASE OF THE NASAL ACCESSORY SINUSES WITH A REVIEW OF ELEVEN CASES*

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INTRODUCTION. — Charity patients often seek relief from disease late, or even very late, so that an early diagnosis cannot be made. Pain and hemorrhage are usually late symptoms of malignant disease. Therefore, the milder symptoms do not attract the patient's attention early as they should. Modern preventive medicine and surgery cannot function for ignorant people very often. This is particularly true of patients admitted to Colorado General Hospital, with and without malignant disease, as most of these patients come from isolated sections of the state. Houser[1] says, "the prognosis varies inversely with the ease of diagnosis." A late diagnosis of malignancy usually means a poor prognosis. The members of the staff are, therefore, forced to give these unfortunates the best and most humane treatment the circumstances will permit. This sometimes means little more than palliative measures. The nearness of the tumor, in the sinuses, to such important structures as the meninges, brain, orbit, optic chiasm, etc., with involvement of bony structures, makes the saving of life very difficult. This leaves much to be desired. Lederer[2] believes "that a malignant neoplasm of a nasal sinus is seldom, if ever, confined within a mathematically calculable area. By virtue of the characteristically slow growth within a bony cavity it is observed as such at a time when it falls within the classification of inoperability; that is, when the diagnosis finally becomes self evident."

This is a relatively small series of cases, but it represents all those of proved malignancy of the sinuses cared for in the institution from September, 1924, to the close of 1936, inclusive.

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Phillips[3] in 1898 called attention to the importance of malignant tumors of the sinuses. There had been occasional reports before that, but his report emphasized the importance of malignancy. He reported one case and collected sixteen others from the literature.

AGE. — The *ages* range from 28, the youngest, to 68, the oldest (28, 39, 46, 47, 48, 50, 56, 62, 62, 67, and 68). The average age was 52+. In one of New's[4] series, reported in 1926, the ages ranged from 3 to 77 years with 50 as the average age. Seventy-nine per cent of his patients were between 40 and 70.

OCCUPATION. — The occupations were as follows: Five were farmers, two were laborers, one was a miner, two were railroad men and one was a housewife. Occupation seems to be of very little importance in malignant disease of the sinuses, although it is important in malignancy of other organs, as one of us (Spencer[5]) has previously mentioned.

SEX. — There were ten males and one female. In one of New's[4] reports, 58 per cent of the primary malignant tumors of the maxillary sinus were in males.

In most of the series of case reports of malignant disease of the head and neck appearing in our literature, males are affected more frequently than females. In the report made by one of us (Spencer[5]) last year of fourteen cases of carcinoma of the tongue, from the University of Colorado General Hospital, thirteen were males.

RACE. — There were nine white Americans and two Mexicans. Our reports of malignant disease nearly always include Mexicans, because of the large numbers of them employed in the sugar beet fields of Colorado.

ONSET. — The onset of the disease preceded admission to the hospital by one month in the shortest case to seven years in the longest. In the patients with a highly malignant type of tumor the duration of life, after the first onset of symptoms, is usually only one or two years.

GLANDULAR ENLARGEMENT. — The superficial cervical glands were enlarged on one or both sides in at least six of the eleven patients. In the other five the deep cervical glands were almost certainly involved at the time of admission, because the sinuses drain

into the deep cervical lymphatics, too, and the disease had lasted long enough, at the time of admission to the hospital, for these to have become involved.

Serological Tests. — The blood Wassermann was negative in all. Other serological tests were made, such as the spinal fluid Wassermann and colloidal gold curve, for a few of the patients and these were negative.

Sinuses Involved. — In seven of the patients the maxillary sinus was first involved. In two it was the frontal, in one the ethmoidal and in one the maxillary, ethmoidal, sphenoidal, orbit and hard palate on one side. The sphenoidal sinus was not primarily involved in any, but it was by extension in two.

Davison and Kahr[6] have reported two cases of primary carcinoma of the sphenoidal sinuses with extension, in both patients, to the cranial cavity. Both were inoperable, but the pain and rapid extension were relieved by irradiation. The autopsies showed the extensive invasion of the cranial cavity just prior to death.

Invasion of Other Parts of the Skull. — The malignancy invaded the orbit in six cases, the hard palate in five, the zygoma in one, the sphenoidal sinus in two and the ethmoidal in one.

Myerson[7] has reported a case in which the right orbit, right ethmoidal, maxillary and sphenoidal sinuses were all involved by carcinoma. The tumor, including the right eyeball, was removed by surgical diathermy with a very good result.

Fenton[8] has emphasized the importance of early recognition of invasion of other parts of the skull, such as the orbit, optic chiasm, meninges, cheek, oral cavity and cranial cavity.

Exophthalmos was present in several of our cases at the time of admission, because the floor of the orbit was involved. Irradiation had apparently not increased the exophthalmos. Schall[9] read a paper dealing with this symptom before the last ear, nose and throat section meeting of the A.M.A. He referred to a recent article by Hill,[10] discussed by Goldsmith, in which the prominence of the eyeball following irradiation was called to our attention. He believes the exophthalmos was made worse by irradiation.

Roentgen-Ray Diagnosis. — The diagnosis was supplemented by x-ray films in all except one. Even when the diagnosis is very evident the x-ray often helps to show extension to other sinuses, the orbit, cranial cavity, optic chiasm, oral cavity, nasopharynx, etc.

METASTASIS. — Other organs of the body were not demonstrably invaded by metastasis, at the time of admission, in a single case by physical examination and x-rays.

TYPE OF TUMOR. — Squamous cell carcinoma is the most frequent type of carcinoma in the sinuses. Bryan[11] has reported a papillary type of carcinoma.

Our ninth case at first suggested clinically that the tumor might be sarcoma, but the pathological report was squamous cell carcinoma. Stone[12] believes sarcoma is more frequently encountered in the maxillary sinus than carcinoma. This has not been our experience nor do the numerous reports in the literature substantiate this.

Hartz[13] mentions the similarity of onset of carcinoma and sarcoma in the maxillary sinus. He says, "if it arises from the floor there are dental signs first, such as neuralgia about the teeth. If it arises in the ethmoidal cells or the orbital plate there may be swelling around the eye or puffiness over the cheek and this swelling will be more persistent than in sinusitis."

Dentists have often been urged to extract bicuspids and molars from the upper jaw to relieve pain, only to find after the extraction the pain was caused by a malignant tumor of the superior maxilla and not by dental disease.

BIOPSY. — A biopsy was done in eight of the eleven cases. In the remaining three the disease was so far advanced a biopsy was not performed and these latter three were probably all cases of carcinoma. The clinical diagnosis of malignancy of the sinuses is usually not difficult. However, the biopsy is very necessary, especially in early cases, to determine the exact type of malignant tumor.

The method used so much by Dr. Gordon B. New of the Mayo Clinic of doing a biopsy while the patient is on the operating table is excellent. He insists the specimen be carefully guarded to avoid any possible confusion with other specimens. The report from the pathologist enables him to decide what type of surgical procedure is best. This is more logical than doing a biopsy one day and the operation the next. The more malignant the tumor the more thorough the surgical excision must be, if surgery is used, and the more thorough the irradiation must be.

Beck and Guttman[14] "have elaborated a dictum that the biopsy determines the details of treatment."

Beck and Guttman[15] also say, "it is only since the recent collaboration of both the pathologist and the radio histologist that one has been able to gain some information regarding the value of the minute picture of the neoplasm as regards the course that the disease pursues and its response to therapy. It has been found that cellular types of neoplasms, which clinically are so highly malignant, and metastasize so readily and extensively, are very frequently the most radiosensitive. For instance, it has been found that neoplasms that are undifferentiated in character, so-called anaplastic lesions, that are highly cellular, and show no tendency towards adult structure, are the ones that are most easily influenced by radio therapy, whether radium or x-ray. The lesions that show a tendency towards adult characteristics — for instance, the acanthotic lesions of a squamous cell carcinoma, that show pearl formations and regular pavement-like tissue structure — respond very poorly to irradiation. These are best handled by extensive surgical destruction."

The final diagnosis usually rests on the biopsy. The macroscopic appearance may be studied at the time of the examination, in the operating room and at the autopsy.

Quick[16] in 1930 offered the following suggestions: "(1) Surgical exploration and biopsy should be done earlier for early diagnosis. (2) Surgical principles in treating new growths elsewhere do not apply to the sinuses, as a rule. (3) Radium and roentgen radiation are of value and should be used in conjunction with surgical treatment. (4) Radium and roentgen radiation, if used in sufficient dosage, can be depended upon to eradicate the lesion. (5) Surgical intervention must be used to secure exposure and drainage. (6) The anatomy of the parts makes infection more of a hazard than elsewhere."

Operable or Inoperable. — Six of the ten were regarded as inoperable at the time of admission. One refused operation. Two had a Caldwell-Luc operation and two a Barnes[17] operation as suggested by Dr. Harry A. Barnes of Boston.

The English otolaryngologists were prone to use surgery alone without irradiation according to a discussion of this subject in 1922 at the March meeting of the Laryngological Section of the Royal Society of Medicine according to Woodman.[18] However, their attitude has changed since 1922, as shown by their discussion in 1931 at a meeting of the same society.

The Caldwell-Luc operation or the Denker may be sufficient for early malignancy of the maxillary sinus. The modified Lynch operation, the Moure, the Barnes, the Preysing or the Kocher may be required for other patients. The operation which will best enable the surgeon to expose and remove the malignant tumor is the one of choice. Even then the operative technique must be altered from time to time to meet the emergencies of the case.

Davis[19] has reported the results from 39 cases during a five year period. He followed many of the cases. Of those followed, thirteen had epitheliomata, four round cell sarcomata, seven endotheliomata, and two spindle-cell sarcomata. The others could not be followed.

Thompson[20] has reported a series of cases in which surgery followed by deep roentgen-ray therapy and radium combined gave better results than any one method of treatment alone.

Nash[21] recommends the cautery, surgical extirpation, endothermy, x-rays and radium in the treatment of malignancies of the sinuses and he employed them in any combinations that were possible at one particular time. He has reported his results in nine cases during a three and one-half year period. He reported five patients dead and a sixth not expected to live. A seventh had a slow-growing sarcoma; the eighth and ninth were cured.

Shutes[22] reported a patient cured after five years. He removed, by means of a Caldwell-Luc operation, a papillary squamous cell carcinoma from the left side of the nose and the left maxillary sinus. This was followed by desiccation with surgical diathermy and by irradiation with radium and the roentgen-ray.

Irradiation. — All received at least some irradiation from either radium or the roentgen-ray except two. Many had extensive irradiation, some before admission, during their stay in Colorado General Hospital and after leaving the hospital. The radiologist at Colorado General Hospital often has to work at a great disadvantage, because he does not know, and cannot always learn, how much irradiation the patient had received before admission. Some of the older roentgen-ray machines do not measure dosage accurately. Our radiologist must often use too little therapy in order to avoid burns and the severe criticism which is so likely to be provoked by burns.

New[4] says, "the combination of cautery and radium, as employed in the Mayo Clinic for the treatment of malignant tumors of the

antrum of Highmore, has eliminated the operative deaths usually associated with this disease, and has produced a higher percentage of cures than previous operative measures."

Greene[23] and Barnes[24] have reported the good results of the combined operative and radium treatment of malignant tumors of the maxillary sinuses. Greene reported eighty-four cases of carcinomata treated at Huntington Memorial Hospital.

Radium in substance, radium emanations and particularly the radium bomb or teleradium of four grams of radium used from different angles has greatly improved radium therapy. We do not have enough radium for a bomb at the University of Colorado General Hospital. Hill[10] has recently mentioned this difficulty in the management of malignant disease of the sinuses as well as others. Only a few large metropolitan areas have radium bombs.

Deep roentgen-ray therapy is another big improvement in irradiation. The 900,000-volt Coolidge x-ray tube insures penetration of the sinuses.

Öhngren[25] favors the combined use of endothermy and irradiation rather than irradiation alone.

Johnson[26] in 1924, in a series of twenty-four cases, used radium alone without surgery or the roentgen-ray. He inserted the radium directly into the sinus and used some on the face and neck surfaces. Four of his patients showed no clinical evidence of malignancy for from one to five years afterward. Three were free from the disease one, three and four years, respectively. One was free from the disease for six and one-half years, but the disease recurred later. Eight patients improved while under treatment, but were not free from the disease at any time. The remaining eight patients lived four years.

Robinson[27] reported ten cases in 1933 treated with radium all well for from two to fourteen years without recurrence.

McCoy[28] reported a case of sarcoma of the maxillary, ethmoidal and sphenoidal sinuses on one side treated with 25 mg. of radium screened with 1 mm. of brass and placed in the antrum and 25 mg. of radium screened with 1 mm. of brass placed in the ethmoidal and sphenoidal region after the sarcoma had been surgically removed. After three treatments, over a period of one month, the pain, swelling and hemorrhage had all disappeared.

Syphilitic glossitis often precedes carcinoma of the tongue and the chronic irritation of the glossitis favors malignancy. In the sinuses, syphilis seems to play a less important role.

In a case previously reported by one of us (Spencer[29]), of primary cholesteatoma of the sinuses and orbit, the chronic irritation finally resulted in a squamous cell carcinoma of the right maxillary sinus, right orbit and right frontal.

Chronic granulomata of the nose and sinuses often produce enough irritation to excite malignant disease as shown by one of us (Spencer[30]) in the Jackson and Coates text book, *The Nose, Throat and Ear and Their Diseases*, published by W. B. Saunders Co., Phila., 1929, pp. 186-197.

Harmer[31] has reported 23 cases. Of these, 18 had carcinomata, all treated surgically with only two deaths soon after operation. Only two patients with carcinomata treated by excision of the tumor were living, without recurrence, after three years. He used excision followed by diathermy and radium.

New[32] in 1933 reported one case of squamous cell epithelioma living after ten years and without recurrence.

New and Cabot[33] in 1935 reported their results in 295 patients with malignant tumor of the antrum and upper jaw. They emphasize the importance of using surgical diathermy, the cautery and radium. They show better results from this method of handling their cases than was previously obtained by surgical removal of the upper jaw, as many patients die following the latter operation.

New and Cabot[33] mention Öhngren's work, as we have done, and they have the following to say about it: "The literature on this subject recently has been very carefully reviewed in a monograph by Öhngren. He traced the development of electrosurgery and irradiation in the treatment of the condition on the Holmgren service of the Sabbatsberg Clinic at Stockholm. At first, the 'soldering iron', or 'ferrum candens', was used; large electric cauteries were then developed; and, finally, electrosurgery. Öhngren reviewed both the foreign and American literature, and his monograph is an outstanding contribution to the subject. He has evolved a clinical classification of his own for determining malignancy of the tumor, in which he combines the position of the growth, with regard to whether indications for operations are favorable or unfavorable, its activity, determined microscopically, and the presence or absence of metastasis. He divides the skull, by means of an imaginary plane passing through the inner canthi and angle of the mandible, into an anterior-inferior or less dangerous portion and into a posterior-superior or more dangerous portion. In turn, each of these portions is further divided into four regions, two lateral and

two medial. Tumors in the medial posterior-superior regions are much more dangerous and treatment is more likely to result in a smaller percentage of cures than with those in the lateral anterior-inferior regions."

Many unimportant portions of the history, negative physical findings and negative laboratory tests have purposely been omitted.

CASE 1. — J.E.B., male, age 48, married, occupation miner, admitted March 31, 1926, and discharged April 16, 1926, as unimproved. The diagnosis on admission was carcinoma of the right maxillary sinus.

His chief complaint was of swelling and pain in the right side of the face.

His illness began in July 1925 with an abscess in the upper gum on the right near the corner of the mouth. This gradually became worse, so that by the following September he had to quit work because he was so weak. In October 1925 he had an operation in another hospital for the removal of the swelling of the gum over the right maxilla. In spite of this operation he grew worse. In February 1926 he had a second operation, at the same hospital, and this time some bone was removed from the right maxilla. Just before the second operation he had two roentgen-ray treatments. His condition became more serious during the five days prior to admission to Colorado General Hospital; the pain was more severe; and, he was unable to open his right eye.

At the physical examination the right eyelids were closed. No inflammation was found when the lids were opened. The right pupil reacted to light. The left pupil reacted to light and accommodation. The right side of the face was swollen, immobile and injected. The patient was unable to open the mouth wide enough for a satisfactory examination. The teeth had all been extracted. The glands in the right side of the neck were markedly enlarged.

The ear, nose and throat consultant reported the nose negative. The nasopharynx was too sensitive to examine. The tonsils were small and infected. A portion of the right superior alveolar process had been removed surgically, exposing the maxillary sinus filled with a sloughing tumor mass with a very bad odor. This was probably carcinoma. The right upper lid could not be opened. The vision was about equal in the two eyes after forcing the right lids apart. The right pupil was smaller than the left and there was exophthalmos of O.D. The movement of O.D. was greatly restricted.

The general surgical consultant recommended codein at night for relief of pain and restlessness. He advised radiation.

The chief of the ear, nose and throat service believed the case inoperable, but suggested a trial with radium. A biopsy was not performed.

The radiologist reported marked fibrosis involving both hili and the whole bronchial system, but no x-ray evidence of tumor metastasis. There was a large rounded shadow of almost homogeneous density occupying the region of the right maxillary sinus and floor of the orbit. The right zygomatic bone appeared thin and partially destroyed. His opinion favored malignant tumor of the right antrum.

The laboratory reported the blood Wassermann negative. The urinalysis was negative. The red blood cells numbered 4,940,000 and the leukocytes 12,200.

The final diagnosis was carcinoma of the right maxillary sinus and orbit.

The patient's progress was not very satisfactory. He was discharged April 16, 1926, as unimproved. The final outcome is not known as the social service department cannot trace him.

CASE 2. — C.C., white, male, age 50, married, occupation farmer, admitted March 20, 1931, and discharged March 27, 1931, as improved. The admission diagnosis was carcinoma of the frontal bone.

His chief complaint was of a growth on the forehead and of frequent sore throat.

His present illness started at the age of 17 with a small lump in the middle of the forehead. This slowly increased in size until seven years ago, it was about the size of a nickel and twice as thick, when it was treated with radium by a dermatologist. About two years ago there was a slight recurrence. At the time of admission there were nodular areas completely surrounding the old scar.

The physical examination showed a well developed and well nourished appearing man somewhat younger than his stated age. The nose was negative. The pupils were round and equal. The reactions to light and accommodation were normal. The eye movements were normal.

In the frontal area, just superior to the nasal bones, and slightly to the right of the midline, in the region of the right frontal sinus, there was an old scar of the skin and subcutaneous tissues. This was adherent to the bone. Surrounding the scar there was a

nodular subcutaneous growth most marked to the right of the growth and superiorly. It extended laterally 2 or 3 cm. The area was about three times the normal thickness. There was slight tenderness to manipulation in this area.

The tongue protruded in the midline and presented a slight peripheral tremor. The gums were injected at the margins. The teeth were poorly cared for. The tonsils were enucleated. The posterior pharyngeal wall was congested and granular. The blood Wassermann was negative. The urinalysis was negative. The red blood cells numbered 5,060,000 and the leukocytes 10,100. A biopsy was not performed.

The provisional diagnosis was carcinoma and probably squamous cell; also sinusitis of the right frontal.

Fifty mg. of radium, screened with 2 mm. of lead, at a distance of 1 cm., was applied on the 25th and 26th of March for sixteen hours each day. On the 27th the same radiation was used for six hours. He was discharged on the 27th to await results. The social service department has not been able to trace him.

Case 3. — E.H., Mexican, male, age 47, married, laborer, was admitted on September 6, 1931, with a diagnosis of carcinoma of nasal polypi. He was discharged Sept. 24, 1931, as unimproved.

His chief complaint was of nasal obstruction and nasal hemorrhages.

In April 1931 he had an attack of influenza characterized by severe headache, generalized aching, malaise, fever, etc. Following this he was unable to breathe through the right side of the nose. A physician gave him a nasal spray and the right side became all right; then the left side closed. There was apparently a good deal of discharge from the nose and in May he began to blow big clots from his nose. His breathing through the nose improved for a while. The hemorrhages became worse. A physician found the left side of the nose closed by a mass of tissue. A biopsy was performed and the tissue sent to the University of Colorado School of Medicine. The pathologist reported squamous cell carcinoma of nasal polypi. Shortly before admission he had a little pain over the left frontal region and in the left orbital cavity.

The physical examination showed a very well nourished Mexican of powerful physique sitting quietly in the ward in no apparent distress. The pupils were equal and regular. Both reacted to light and accommodation. The eye movements were normal. There was no nystagmus or strabismus. The scleræ and conjunctivæ were

negative. The left nasal fossa was completely obstructed and the right partially so. There was a mucoid, bloody secretion coming from the nose. There was a white, glistening mass of tissue in the left side which bled freely upon manipulation. The left frontal and maxillary sinuses were dark by transillumination. The teeth were in poor condition. Many had been extracted. The gums were retracted, discolored and infected. The mouth was filthy and the breath foul. The tongue protruded in the midline and there was no tremor. The tonsils were of normal size and apparently chronically infected, because the blood vessels were injected and the anterior pillars red.

The provisional diagnosis was carcinoma of the nasal accessory sinuses on the left.

On September 15, 1931, there was an abscess of the left cheek which was opened under gas anesthesia and foul smelling pus evacuated on the 17th. He was much better on the 18th.

The radiologist reported all sinuses on the left, except the frontal, homogeneously opaque, probably due to tumor invasion.

The blood Wassermann was negative. The urinalysis and blood count were negative.

The patient's condition was considered inoperable. He was given deep roentgen-ray therapy on September 9, 11, 13 and 19. He was discharged on the 24th as unimproved. The social service department has not been able to follow this patient.

A biopsy specimen taken from the nose consisted of a mass of soft gray tissue 1 cm. in diameter. Microscopic examination of this revealed an edematous, fibrous stroma resembling that of a nasal polyp, invaded by groups of acidophilic polyhedral and pyramidal shaped epithelial cells. A tendency of these cells to form small clusters was seen. The nuclei were large and finely reticular. Mitotic figures were numerous. The cell groups were suggestive of neither glandular nor pavement arrangement, and none of the characteristics of squamous epithelium, such as keratohyalin granules, epithelial pearl formation, or spinous processes were seen. The growth bears a resemblance to squamous cell carcinoma, but was distinguished by the absence of the above mentioned features. The surface was partly covered by an atrophic layer of pseudostratified columnar epithelium, changing to thin stratified squamous. There was extensive ulceration and suppurative inflammation near the surface. Where a surface layer of epithelium was present it had no evident relationship to the tumor cells. The tumor repre-

sented a sub-variety of epidermoid carcinoma, variously known as transitional cell carcinoma, lymphoepithelioma or Schmincke tumor. It originated in mucous membranes with a stratified squamous epithelium, frequently that covering lymphoid tissue, as in the nasopharynx. The primary growth may be small, while secondary growths in the lymph drainage area are bulky. It may, on this account, be confused with branchiogenic carcinoma or with a primary tumor of the cervical lymph nodes.

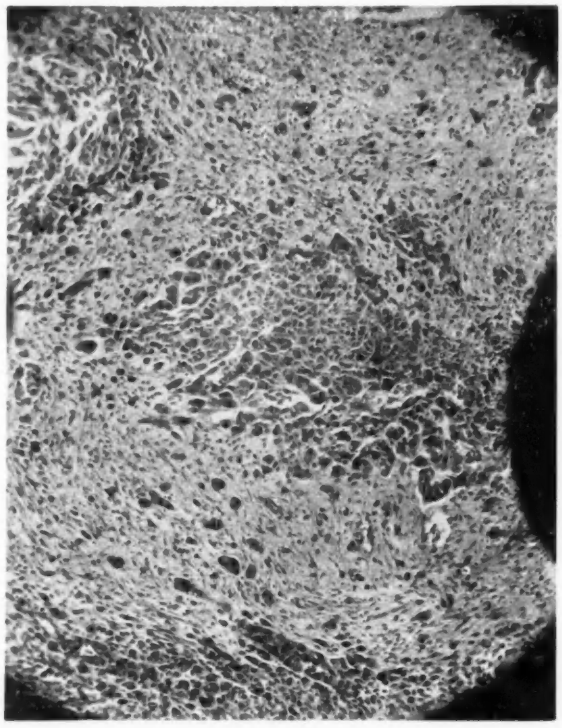

FIG. 1 — (Spencer and Black) — Case 3. Squamous Cell Carcinoma of left superior maxilla, orbit, ethmoid and sphenoid magnified 75×.

According to Ewing[34] the transitional cell carcinoma of the nasopharynx has "been recognized by many authors, but has never been sufficiently emphasized. It should be noted that surgical results in this type of epidermoid carcinoma are poor owing to numerous and widespread metastases, while the undifferentiated character of the cells renders them highly susceptible to radiation. The pri-

mary tumor and the cervical metastases may often be cleared up by heavy external radiation, but most of the cases eventually succumb to internal and distant metastases."

CASE 4. — M.E.A., female, age 62, white, widow, was admitted December 13, 1932, and discharged December 19, 1932. The diagnosis on admission was carcinoma of the left maxillary sinus.

Her chief complaint was of pain, tenderness and swelling of the left cheek.

She had catarrh of the nose for a year before admission, which bothered her only when she had a cold. About three months before admission she noticed the whole left side of her face was swollen, but this was more marked under the eye and over the cheek bone. There had not been constant pain, but the cheek was tender to pressure and to cold. There had been more discharge, with a foul odor, from her left nostril since the swelling started. She had lost eighteen pounds during the three months before admission.

The physical examination revealed a poorly nourished white woman, with a dark skin and of stated age, lying in bed quietly with a swelling under the left eye. There was a swelling over the left maxillary sinus with some serous discharge at a point inferior to the outer canthus. The left eyelids were partly closed due to the edema from below. The pupils were equal and regular and reacted to light and accommodation. The left side of the nose was partially occluded due to the lateral pressure. The throat was negative. There was some cervical adenopathy.

The blood Wassermann and urinalysis were negative. The red blood cells were 3,760,000. The leukocytes were 6,450.

The provisional diagnosis was malignancy of the left maxillary sinus.

A biopsy was performed and showed a squamous cell carcinoma. She was given her choice of a radical operation or deep roentgen-ray therapy after the disease had been explained to her. She elected to have deep roentgen-ray therapy. She was discharged to the out-patient department as unimproved for this treatment.

The tissue received was a biopsy of the left antrum, and consisted of fragments of firm, pale tissue. Microscopically the tissue was composed of broad sheets of large, pale, polyhedral, epithelial cells infiltrating a dense fibrous stroma in which there were large numbers of lymphocytes, plasma cells and eosinophilic leukocytes. The epithelial cells possessed spines, occasionally keratohyalin

granules and formed many epithelial pearls. Mitotic nuclear figures were numerous. The structure was that of a moderately well-differentiated squamous cell carcinoma.

She died in October, 1935.

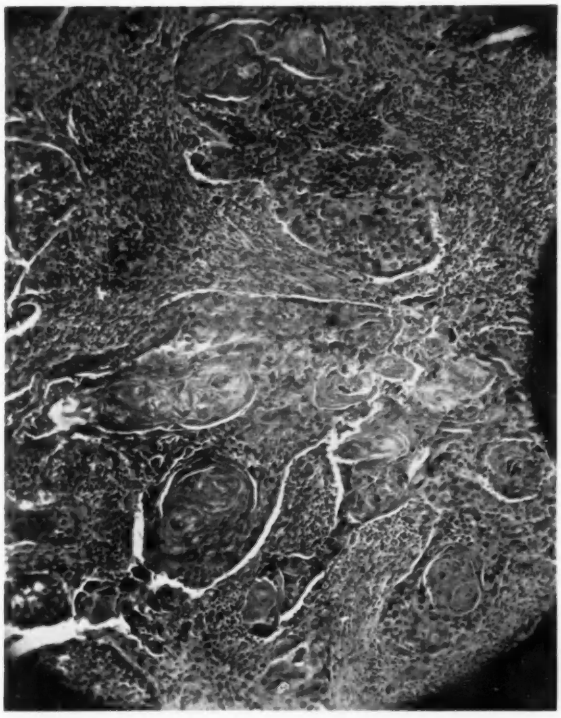

FIG. 2 — (Spencer and Black) — Case 4. Squamous Cell Carcinoma of left maxillary sinus magnified 75×.

CASE 5. — R.B.W., white male, age 28, occupation railroad brakeman, single, admitted December 19, 1932, and discharged the first time on February 22, 1933. He was readmitted several times during the subsequent six months for reexamination and treatment. He made a complete recovery and is living and well. The diagnosis on admission was tumor of the left maxillary sinus.

His chief complaint was of swelling over the left side of the face and under both eyes. There was some soreness over the right side of the face.

Three years before admission he had a small nodule removed from his right foot and all his upper teeth extracted. Some of the

upper teeth were abscessed. The left maxillary sinus became infected and a fistula formed through the alveolar process. The sinus was frequently washed through this fistula. The sinus infection improved except for the swelling of the left side of the face. His nose had been almost completely obstructed during this three year period. One month before admission he began to have headaches, especially after stooping over. Heat was applied for one week. One week before admission a physician attempted to open the left maxillary sinus through the nose and wash the sinus, without success. Both lower eyelids began to swell five days before admission and the conjunctivæ became bloodshot, but the right more than the left.

The physical examination revealed a well nourished white male, of about the stated age, lying quietly in bed breathing through his mouth and with the face swollen. The left side was worse. The conjunctivæ were injected and the right more than the left. There seemed to be some exophthalmos on the left. The pupils reacted to light and accommodation and were regular and equal. There was complete obstruction of both sides of the nose, and mucous membranes were injected. All of the upper teeth had been extracted and some of the bone had been destroyed on the upper left. The tongue protruded in the midline. The tonsils were out. The pharynx was negative. The cervical and submaxillary glands were enlarged bilaterally. The thyroid could not be palpated. The chest was symmetrical and the expansion equal. There was a mass to the left of the posterior spinous process of the tenth thoracic vertebra. This was nodular, firm and attached to the underlying tissues.

The urinalysis, blood count and blood Wassermann were all negative.

The provisional diagnosis was tumor of the left maxillary sinus probably sarcoma.

A biopsy was performed on December 21, 1932, under local. The tissue showed suppurative inflammation with necrosis.

The radiologist reported the frontal sinuses were absent. There was marked homogeneous cloudiness over the left maxillary sinus and left ethmoidal. The sphenoidal sinuses, the right maxillary and the right ethmoidal were hazy. There was some erosion of bone over the oral aspect of the hard palate on the left, but no definite evidence of osteomyelitic involvement of the bony walls of the sinuses.

On December 31, 1932, a Caldwell-Luc operation was performed on the left maxillary sinus under local. The bony walls were found with cystic material and much chronic inflammatory tissue was removed.

On January 6, 1933, tumors were removed from the back, right arm and forearm under a local anæsthetic.

The patient received a series of deep roentgen-ray treatments over a period of months. He was finally discharged as symptom free. He is living and well almost at the end of the five year period.

FIG. 3 — (Spencer and Black) — Case 5. Photograph of patient, taken three days after admission, with Neurogenic Sarcoma of the left maxillary sinus.

Multiple tumors were present, but the first to cause serious symptoms was located in the left antrum. A biopsy of this tumor was made and later similar tumors were removed from subcutaneous sites on the back and left arm, the latter being attached to the median nerve. A tumor diagnosed elsewhere as a fibroma had

been previously removed from the tendon of the right great toe. A subcutaneous tumor of the right thigh appeared between the time of the patient's first and second admissions to this hospital, which was not removed, but was treated by x-ray.

Aside from inflammatory changes present in the antral tumor, all were alike in structure. They were composed of small spindle shaped cells with abundant fibrillar intercellular substance containing fairly numerous thin walled blood vessels. The cells were in bundles cut in many planes, and where these were cut longitudinally a tendency to parallel arrangement with palisading of the nuclei was seen. This arrangement was nowhere well defined, and in many areas was completely lacking. The nuclei varied greatly in size, shape and staining quality, but mitotic nuclear figures were not found.

In certain areas the intercellular substance was vacuolated, and such sections stained for fat showed the presence of small fat droplets. The amount of intercellular substance varied in different parts, and in some locations the stroma was fibrous and partly hyalin.

In sections of the tumor of the antrum, hemorrhage, necrosis and leukocytic infiltration were prominent, and no capsule was evident. In the tumors of the back and arm, a heavy fibrous capsule was present.

The histologic structure of these tumors was consistent with that of neurogenic (neuro) sarcoma. Stewart and Copeland[35] point out that many sarcomas of soft parts are neurogenic and are similar both clinically and histologically to the lesions of Von Recklinghausen's disease. These may frequently be associated with one or more of the stigmata of this condition. In this patient, multiplicity of the tumors was suggestive of neurofibromatosis, but skin pigmentation was not noted, and no family history of the disease was recorded. The location of one of multiple tumors in the antrum was incidental in this case, as such tumors may occur anywhere in the body.

Recurrence after excision is common in neurosarcoma. The effect of irradiation in preventing or delaying recurrence following excision is not clearly defined. In general, prognosis is poor. The tumors vary in degree of malignancy, from slow growing or stationary fibrous encapsulated tumors to rapidly growing infiltrating and metastasizing types.

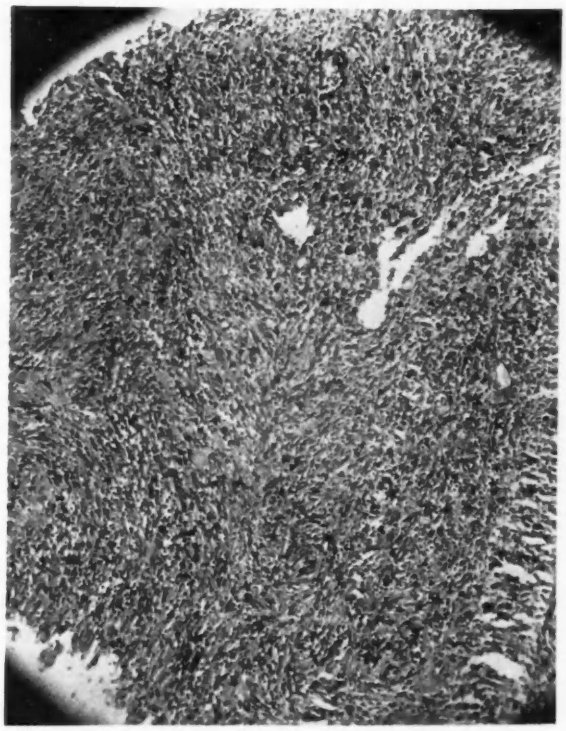

FIG. 4 — (Spencer and Black) — Neurogenic Sarcoma of left maxillary sinus, orbit, hard and soft palate magnified 75×.

In this case no recurrence of the antral or arm growths had been observed within 6½ months after removal. The tumor of the thigh had not increased in size following irradiation.

CASE 6. — W.O.R., white, male, age 68, occupation farmer, married, admitted October 17, 1933, and discharged December 13, 1933, as improved. The diagnosis on admission was malignant disease of the left maxillary sinus.

His chief complaint was of a burning sensation and swelling of the left side of the face with constant headache in the region of the forehead.

One year before admission he began to have rather severe generalized headaches, especially upon arising in the morning. Six months later the headaches become more severe and localized between the eyes and just above the eyebrows. This was especially

severe upon stooping over. He consulted his physician who told him he had sinus trouble and did an operation for the removal of "some bones." Two days later the surgeon cut something open in the side of his nose. Following this his face began to swell; the headaches became more continuous; and his hearing became less acute on the left side. The surgeon then sent him to Colorado General Hospital. He had lost fourteen pounds since the first operation was performed.

The physical examination showed a normally developed, rather poorly nourished, white male, walking about, complaining of pain and swelling of the left cheek. The conjunctivæ were normal. The corneæ were normal. The left eye watered rather profusely. The pupils were regular and equal. They reacted sluggishly to light and accommodation. There were crusts in the anterior naris on the right. There was profuse mucoid secretion on the left. All the nasal mucous membranes were congested and swollen, but worse on the left. The left cheek, over the maxillary sinus, was swollen, red and tender. Many of his teeth had been extracted; those he had were badly decayed. There were many cavities and old fillings. The gums had receded. The tongue was clean and it protruded in the midline. The tonsils were atrophic.

The radiologist reported the left frontal sinus hazy and the left maxillary, ethmoidal and sphenoidal markedly cloudy. The urinalysis, blood count and blood Wassermann were all negative.

On October 21, 1933, a Caldwell-Luc operation was performed with the removal of a soft tumor which filled the left maxillary sinus. A similar tumor was removed from the left middle meatus. A frozen section was positive for malignancy and probably carcinoma.

On November 8, 1933, an extensive external rhinotomy was performed with evacuation of all of the sinuses on the left side except the frontal.

On November 11, 1933, radium was placed near the posterior wall of the left maxillary sinus. The 50 mg. were screened with 1 mm. of brass and 2 mm. of lead at a distance of 1 cm. and left for 48 hours.

On November 24, 1933, the same radiation was used for 38 hours under the skin of the anterior maxillary wall. On December 3, 1933, the patient's condition was greatly improved and the wound was healing satisfactorily. The patient was discharged on December 13, 1933, in a very satisfactory condition.

The final diagnosis was plasmacytoma.

Plasmacytoma of a sinus is apparently rather rare. However, Walgren[36] has reported one hundred and twenty-five occurring in the long bones, of which thirty-five were of a definite plasma cell type.

The patient was readmitted on July 17, 1934, for reexamination, observation and treatment. There was no roentgen-ray evidence of metastasis to the osseous system. There was no evidence of recurrence. The external wound was closed under local. Tumors of the right cubital fossa were removed under local also. His

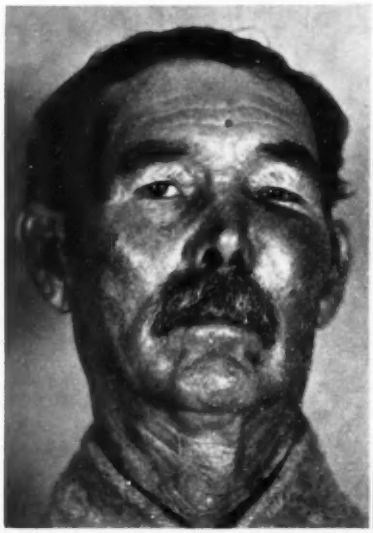

FIG. 5 — (Spencer and Black) — Case 6. Photograph taken two weeks after admission and just before operation for Plasmacytoma.

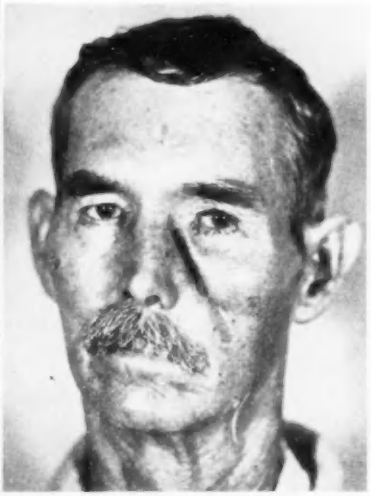

FIG. 6 — (Spencer and Black) — Case 6. Photograph taken one month after radical operation for removal of Plasmacytoma.

general condition was not very good and the prognosis was poor. He was discharged on August 11, 1934. The social service department has not been able to follow this patient.

This case has been previously reported by Dr. Kemp G. Cooper[37] as a solitary plasmacytoma. Since his report new tumors have occurred. The biopsy specimen consisted of a number of small soft white pieces of watery tissue from the antrum and nose. The operative specimen consisted of several pieces of fairly firm whitish-gray tissue, the largest measuring 3x2x0.5 cm. Following

the operation and subsequent irradiation the patient was discharged as improved, to return for observation and further radiation therapy. On his return in two months there was no evidence of recurrence at the operative site, but two tumors, one of walnut size, the other of pea size, of one month's duration, were present beneath the skin of the right cubital fossa. These tumors were removed surgically. Grossly the larger was firm, pink, spheroidal, smooth on one side, rough on the other with tabs of adipose tissue attached.

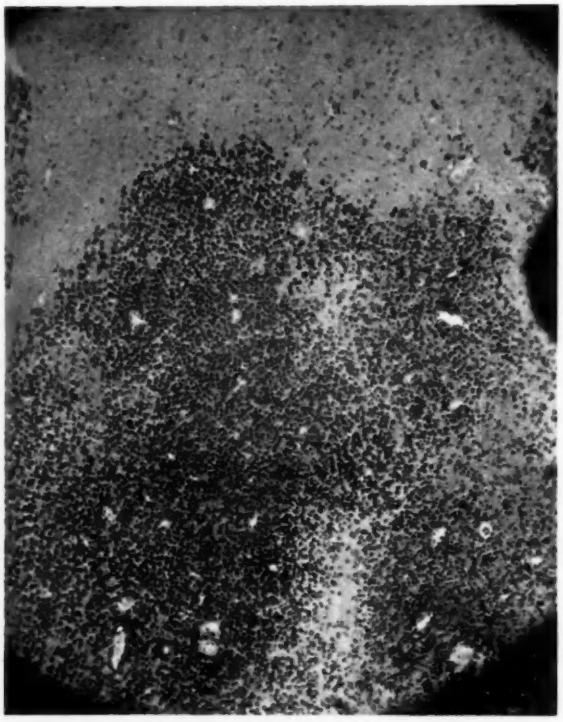

FIG. 7 — (Spencer and Black) — Case 6. Plasmacytoma of left maxillary sinus magnified 75×.

Microscopically, all tumor tissue from various sites, including the nose, antrum and arm, was alike and was composed of solidly grouped large, round or polygonal cells with violet staining cytoplasm and deeply basophilic eccentrically placed nuclei. Double nuclei and mitotic nuclei were frequently seen.

In the tumor tissue from the nose and antrum the surface was partly covered by pseudostratified columnar epithelium, and the tumor cells were in solid masses in the edematous lamina propria beneath it. In the arm tumor the cells formed a solid non-encapsulated mass lying in a fibrous and adipose tissue stroma. The close resemblance of these cells to plasma cells was apparent, and was very evident in pyronin-methyl green stained sections, where the characteristic polychrome reaction was seen. The diagnosis of malignant plasmacytoma was made upon clear cut histological evidence.

No evidence, either x-ray or clinical, of bone involvement existed in this case, distinguishing it from either multiple or solitary myeloma of bone. The arm tumors were probably metastatic but may have represented multicentric foci. The clinical and pathological features of such tumors has been reviewed by Blumenfeld.[38] It would appear that plasmacytoma, apart from the tumor of bone, is fairly frequently found in the upper respiratory tract. That such tumors are true neoplasms is generally accepted. The cause is unknown. Inflammation may be a predisposing factor. The histologic relationship of solitary extraosseous plasmacytoma to multiple myeloma of bone is apparently very close, but the clinical manifestations are quite different, as noted by Cooper.

Jackson, Parker and Bethea[39] believe that plasma cell tumors belong to the group of malignant lymphomata, and that all, including multiple myeloma, are interrelated, occurring as a result of the same fundamental process.

Case 7. — J.Q., male, age 56, white, married, admitted the first time on Oct. 18, 1934, and discharged the first time on leave January 10, 1935. The diagnosis on admission was neoplasm of the right maxillary sinus. His chief complaint was swelling in his nose.

The patient first noticed trouble in March 1934 when a small lump appeared and his right nostril became so full he could not breathe through it. The area gradually became larger and deeper red in color. By May 1934 it had become large enough to make the right eye swell shut. The area had not been painful or tender. His vision was not affected. He had more discharge from his nose after the mass started to form. Following an operation in August 1934, he was unable to breathe through his nose. There had been some bloody discharge from the left side.

The physical examination revealed a well nourished white male of about 55 years of age, lying quietly in bed complaining of a

swelling on the right side of his face which had closed the right eyelids and the right side of the nose. His left pupil reacted to light and accommodation. The movements of the left eye were normal. On the right he had a typical Horner's syndrome. The area about the eye was red and swollen with some purulent discharge in the palpebral fissure. The pupillary reactions were sluggish and the conjunctiva injected. The left side of the face was normal. On the right there was a hard, firm, nodular mass about the size of a hen's egg, which was very red. The left side of the nose was exter-

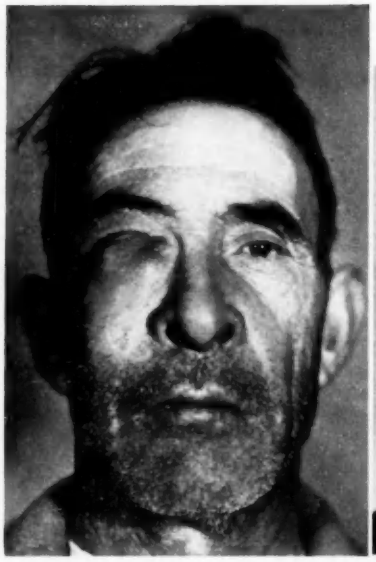

FIG. 8 — (Spencer and Black) — Case 7. Photograph of patient taken four days after first admission to the hospital, with Squamous Cell Carcinoma.

FIG. 9 — (Spencer and Black) — Case 7. Photograph of patient with Squamous Cell Carcinoma of right maxillary sinus taken after three months of treatment.

nally negative. The right side was edematous, red and swollen. It contained a little discharge. A number of teeth had been extracted. Many teeth were carious. The gums had pyorrhœa. The tongue protruded in the midline and there were no tremors. The pharynx was injected and coated.

The provisional diagnosis was carcinoma of the right maxillary sinus.

The radiologist reported both maxillary sinuses were dense. There was extensive destruction of the superior, inferior and lat-

eral walls of the right maxillary. The right ethmoidal cells and the right sphenoidal sinus were cloudy. The right frontal was negative. The left frontal was small and hazy. The urinalysis, blood count and blood Wassermann were all negative.

On October 24, 1934, the patient had cancerous material removed from the right maxillary sinus, with a ronguer and curette, under ether anesthesia, by the Caldwell-Luc incision. The pathologist reported a very malignant squamous cell carcinoma. The same day the patient received deep roentgen-ray therapy. A radium pack of 50 mg., screened with 2 mm. of lead and 1 mm. of brass, was inserted through the Caldwell-Luc incision. This was left in place for 72 hours.

On October 26, 1934, an area of skin broke down near the nose on the right. This exposed the carcinoma. The ophthalmologist who saw him could not find any orbital involvement.

He was discharged on leave November 11, 1934, to return for more treatment, but he did not return until the pain became unbearable.

He was readmitted on September 16, 1936, with a generalized carcinoma of the entire right side of the face. The cervical glands on the right were extensively involved, but the radiologist did not believe the lungs showed evidence of metastasis. There was a large, warty, fungating mass involving the right side of the face, extending into and above the right orbit, compressing the right nasal cavity and extending into the right side of the mouth. The breath was foul and the patient coughed a good deal. The chest examination revealed slight flatness of the percussion note throughout with diminution of the breath sounds. The chief of the ear, nose and throat service considered further treatment useless. Symptomatic treatment was instituted. The patient died in the hospital on October 31, 1936, following a generalized failure of the cardiorespiratory system.

The original biopsy was a small, firm mass of grayish tissue from the antrum. Microscopically there was a loose reticular stroma heavily infiltrated by lymphocytes and polymorphonuclear leukocytes and invaded by coarse bands of polyhedral acidophilic epithelial cells with well defined spinous processes, but without other differential characters. Cells in mitosis were numerous. The tumor was a highly malignant form of squamous cell carcinoma.

This patient's death occurred Oct. 31, 1936, two years after his first admission, from extension of the tumor. Autopsy disclosed involvement of the meninges and brain.

FIG. 10 — (Spencer and Black) — Case 7. Photograph of patient with Squamous Cell Carcinoma of right maxillary sinus taken six weeks before death.

FIG. 11 — (Spencer and Black) — Case 7. Photograph of patient with Squamous Cell Carcinoma of right maxillary sinus taken a few hours after death.

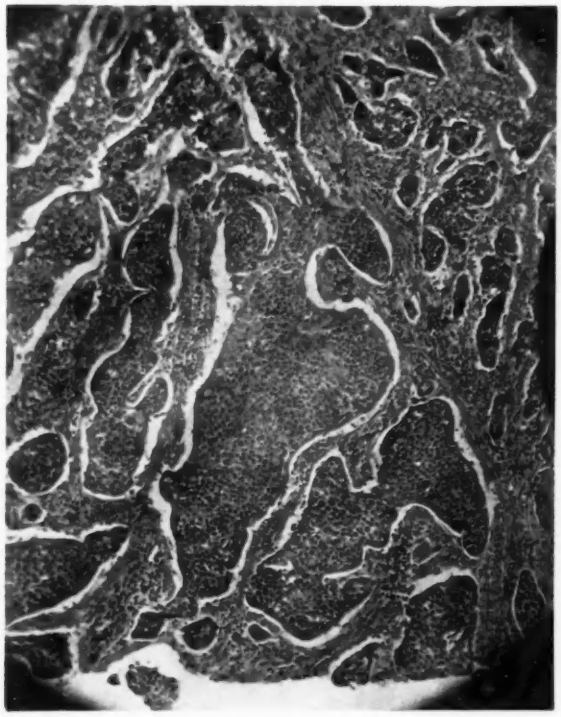

FIG. 12 — (Spencer and Black) — Case 7. Squamous Cell Carcinoma of right maxillary sinus magnified 75×.

CASE 8. — P.J.K., white, male, age 39, single, occupation farmer, was admitted on December 27, 1934, and discharged on January 4, 1935, without treatment. The diagnosis on admission was probable primary carcinoma of the frontal sinus with secondary carcinoma of the brain, lower brain stem. etc.

His chief complaint was of pain in the side of the head.

He had suffered from pain in the right side of his head around the right orbit and in back of his head for six weeks before admission, following what he believed was a sinus infection. He had nausea and vomiting once. On December 21, 1934, several enlarged glands were removed from the right side of the neck. His face was numb over the entire right side, but this was better than it had been. He had not been able to open his right eyelids for two weeks and before that he had diplopia. He was too weak to walk. The pain was becoming so severe he had to have two or three hypodermics each night.

The physical examination revealed a gaunt, well developed male of about 40, lying in bed groaning and apparently in great pain. The skull was very tender over the right supraorbital ridge and along the right side of the nose. The external nose was negative. The right eyelids were closed. The left lids were normal. The conjunctivæ were normal. The pupils were round, equal and regular. They reacted to light and accommodation. The left eyeball moved normally except for moderate horizontal nystagmus. The right eyeball did not move. There was vision in each eye. The movement of the right side of the mouth was limited. The teeth were badly stained and worn down to the gum margins. There was a white deposit at the gum margins. The tongue showed a coarse tremor and protruded to the right. The tonsils were normal in size, but contained pus. The pillars were inflamed. There were hard, enlarged glands in the anterior and posterior chains. The glands were discrete with slight to moderate fixation to the skin and deep fascia. There was a recent surgical scar in the right submaxillary triangle. The area supplied by the right second division of the fifth nerve was very hyperesthetic. The nuclei of the fourth, fifth, sixth and seventh nerves on the right were involved.

The urine contained one plus albumin, one plus pus and two red cells. The white count was 10,550 with 68 per cent polymorphonuclears. The Wassermann and Eagle were negative. The radiologist reported marked cloudiness of both frontal sinuses and of the right sphenoidal. The other sinuses were negative. The spinal fluid pressure was 90 mm. of water. The spinal fluid chemistry showed an increase in the sugar and a decrease in the protein. The salt study, Wassermann and colloidal gold curve tests of the spinal fluid were normal. The staff believed the carcinoma had invaded the brain by metastasis.

The patient was kept quiet with narcotics. He was very irritable and at times irrational. His temperature reached 104 on January 4, 1935. The temperature was irregular. No cause was found for this. His lower lip became swollen on January 4, 1935, and was bright red. There was a large ulcer, covered with a grayish-white exudate on the mucous membrane. This was negative for Vincent's, but it looked like a Vincent's ulcer.

Against the advice of the staff, the patient's family physician removed him from the hospital on January 4, 1935. The patient died three days later.

CASE 9. — A.G., a Mexican, male, age 48, single, laborer, admitted March 27, 1936, and discharged May 28, 1936, on leave to return for more treatment. The diagnosis was malignant disease of the right maxillary sinus on admission.

His chief complaint was of constant pain in the right side of the face and nose. There was intermittent pain in the region of the right eye.

He had not had any trouble with his nose until January 1936 when he thought he had a cold. Late in January he noticed the swelling which gradually became larger. In February 1936 his physician opened an abscess (?) below the right eye. Following the operation he had more pain on the right and more swelling. An open wound had remained since the abscess was opened. He had raised sputum ever since he first noticed the growth, but he didn't have any pain in his chest. He felt as if the sputum came from his nose. It had been bloody occasionally. He had difficulty hearing with the right ear.

The physical examination revealed a fairly well developed, poorly nourished Mexican man of about the stated age of 48. There was a large swelling on the right side of the nose and right maxilla with an open wound on the right maxilla about an inch in length exuding purulent material. The tissues below the right eye were swollen and he had difficulty in opening the right eye. He had vision in the eye. The right side of the nose was entirely occluded by a neoplasm. There was tenderness over the area involved by the swelling. The tongue was clean and the palate of normal contour. The tonsils were small and imbedded. The superior portion of the anterior pillars was injected. The pharynx was normal. There was slight adenopathy.

The urinalysis and the blood Wassermann were negative. The erythrocytes were 3,900,000. The leukocytes were 12,400.

The radiologist reported an almost homogeneous density involving the right maxillary sinus, right nasal fossa and lower portion of the right orbit. He thought the floor of the right orbit had been destroyed by the neoplasm indicating an invasion by a malignant tumor. The left maxillary sinus was cloudy. There was considerable calcification of the pineal gland. There was no roentgen-ray evidence of pulmonary parenchymal, pleural or cardiac pathology.

On April 3, 1936, a biopsy specimen was taken from the right side of the nose and from under the skin of the right malar region.

The bleeding was profuse and was controlled by the electric cautery. The final diagnosis was squamous cell carcinoma of the right maxillary sinus.

On April 6, 1936, he was given a roentgen-ray treatment. He was given three more treatments during the next three days.

Beginning on May 11, 1936, he received three more x-ray treatments over a period of three days.

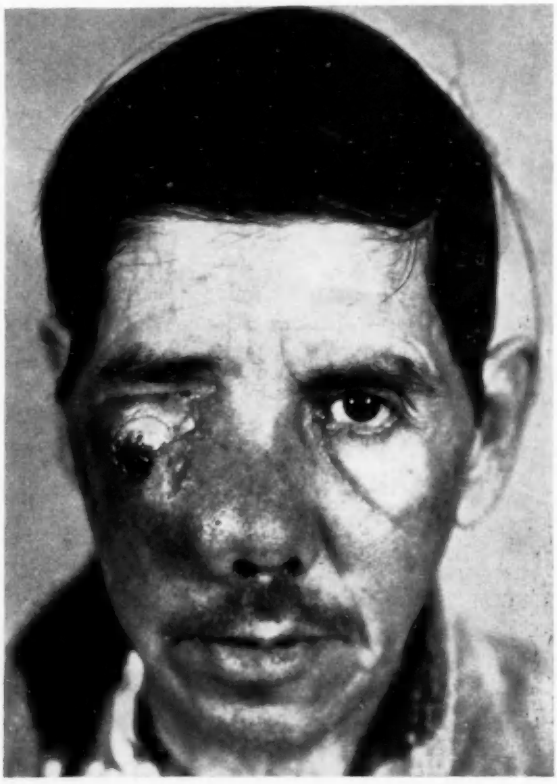

FIG. 13 — (Spencer and Black) — Case 9. Photograph of Mexican with Squamous Cell Carcinoma of right maxillary sinus one week after admission.

On April 13, 1936, under local anesthesia, a large fungating mass was removed from the right side of the nose, so that an opening was made back to the posterior naris. The bleeding was profuse and was controlled by the electric cautery. The tissue was kept to be used as a culture medium for the B. histolyticus. He was

given several treatments with the ensol. He was discharged on leave as improved on May 28, 1936, to return in two months. He did not return. The social service department has not been able to trace him.

The biopsy specimen consisted of two pieces of tissue, one was part of a mass obstructing the right nostril, and the other was tissue removed through a previous skin incision over the zygomatic

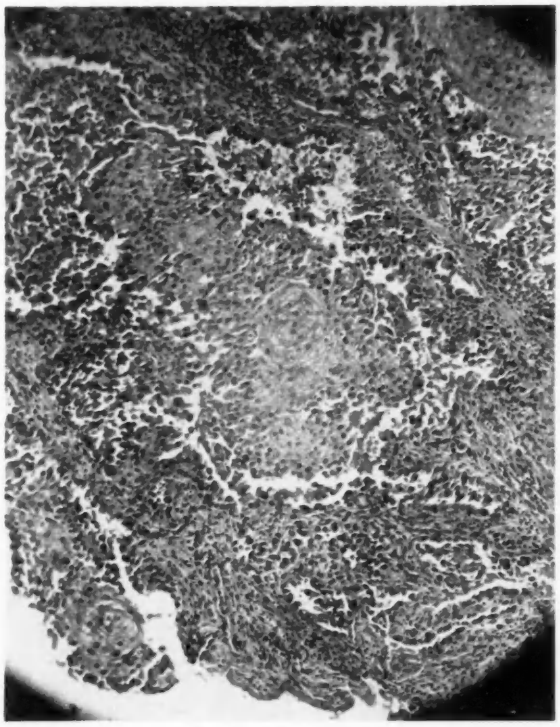

FIG. 14 — (Spencer and Black) — Case 9. Squamous Cell Carcinoma of right maxillary sinus magnified 75X.

prominence of the right maxilla. Microscopically, these were made up of a typical squamous epithelium growing in broad sheets in a fibrous stroma. Epithelial pearls were present, but were not well formed and retained single cell outlines. Keratohyalin granules were lacking. A large part of the tumor was composed of cells undifferentiated except for occasional spinous processes, with a tendency toward papillary arrangement. Mitotic figures were quite

numerous. The tumor was a highly malignant squamous cell carcinoma. The outcome of the case, following x-ray therapy, is not known.

CASE 10. — J.C., male, age 62, farmer, married, admitted Aug. 18, 1936, and discharged Nov. 12, 1936. The diagnosis on admission was malignancy of the right orbit.

His chief complaint on admission was of diplopia.

In March 1936 the patient noticed a lump under the upper right eyelid. Ten days later the lump seemed to travel behind the eyeball and disappear. After several weeks he noticed the vision, O.D., became blurred and he had diplopia. He had lost weight since March, 1936. He believed his eyeball had been pushed forward. There was a sensation of throbbing in the orbit.

The general physical examination revealed a poorly developed, white male, of stated age. His skin was dry and freckled with a few hypertrophic spots over each side of the neck. There was a blood clot in the right side of the nose. There was some exophthalmos on the right with a little chemosis at the canthi. The pupils were equal and reacted to light and accommodation. The eye movements were normal. Several teeth were missing. There was much anterior cervical adenopathy. The thyroid was palpable, but not enlarged. There was some soft tissue retraction bilaterally in the supra- and infra-clavicular regions. There was a small tumor mass in the left lumbar region. This was removed on August 20, 1936. He had hyperkeratosis of the skin below each ear. These masses were removed August 20, 1936.

The patient had diplopia on looking to the right, left or upward and nystagmus when he looked to either the right or left, but more marked to the left. The pupils and the fundi were normal. The movements of the face and tongue were normal. The hearing was normal for forks of low pitch. There was no disturbance of equilibrium and none of coordination. There was no tremor.

A Caldwell-Luc operation was performed on the right side under local anesthesia on September 12, 1936. The mucosa was considerably thickened and polypoid. The roof of the sinus was composed of thin, eggshell-like bone intermixed with tissue which resembled granulation tissue, and which was believed to be carcinomatous from the orbit. This tissue and that from the sinus was sent to the laboratory. A large intranasal window was made in the right inferior meatus. The labiogingival incision was closed with silk sutures.

On September 12, 1936, an incision was also made through the conjunctiva to the periosteum of the orbit. The periosteum was peeled back and bleeding controlled by pressure. A portion of the orbital contents was removed with a snare and the remaining portion dissected out. Along the floor of the orbit there was a mass of tissue which had eroded through the bony orbital floor. This mass involved practically the entire orbital floor, nasal and temporal walls. The mass was removed by electro-coagulation and curettage,

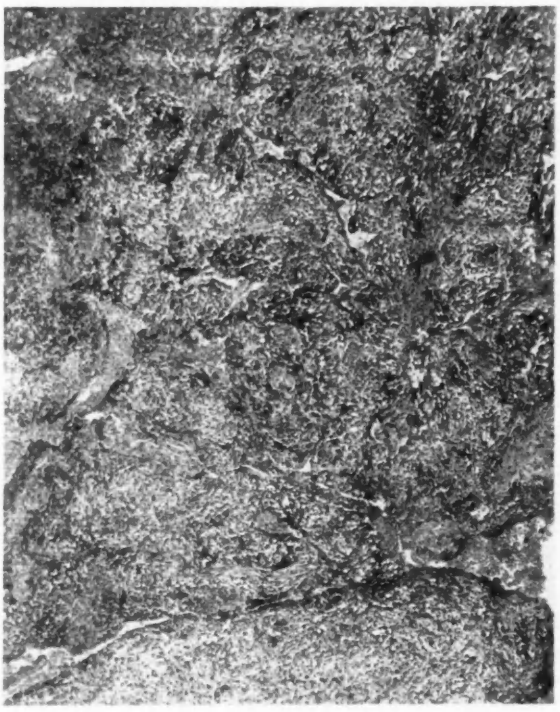

FIG. 15 — (Spencer and Black) — Case 10. Squamous Cell Carcinoma of right maxillary sinus and orbit magnified 75×.

so that a large opening was made from the orbit into the maxillary sinus. The area was electro-coagulated. An iodoform pack, saturated with mineral oil, was inserted into the sinus and through the roof into the orbit. Several silk sutures were used to close the canthotomy.

On September 24, 1936, 45 mgs. of radium were placed in the opening previously made in the orbital floor. Lead sheeting was

placed over the surface of the eyelids. This was left in place for 24 hours.

The radiologist reported there was no evidence of intrathoracic tumor invasion on August 24, 1936. There was some accentuation of the aortic arch. The diaphragm and costophrenic angles were clear.

The urinalysis was negative on Aug. 18, 1936. The erythrocytes were 3,950,600 and the leukocytes 6,600 on the same date. The spinal fluid Wassermann and the colloidal gold curve were negative.

The final diagnosis was squamous cell carcinoma of the right maxillary sinus and orbit. His progress in the hospital was satisfactory. He is living and well.

Two tissue specimens were obtained; one from the antrum and the other from the floor of the orbit. Both were fragmentary masses of pink, soft tissue. Microscopically, in tissue from the antrum, the lamina propria was occupied by large groups of clear polyhedral epithelial cells with numerous mitoses. Differentiation was lacking in these cells. The mucosa was intact with many mucus secreting cells in the pseudostratified columnar epithelium. The stroma was heavily infiltrated with lymphocytes and plasma cells.

In sections from the floor of the orbit, the fibrous tissue was invaded by similar groups of cells in which an occasional epithelial pearl was present, and where spinous cytoplasmic processes were seen.

The diagnosis was squamous cell carcinoma, probably primary in the orbit rather than in the antrum.

Case 11. — W.F.A., age 67, white, male, married, retired, admitted December 27, 1933, and discharged January 24, 1934. The diagnosis on admission was deferred.

His chief complaint was of pain in the left side of his face with swelling. There was more or less constant headache, double vision and impaired hearing on the left.

One month before admission he noticed a small swelling at the corner of the left eye. The eyeball gradually became more prominent. He complained of some headache and a moderate amount of pain. The submaxillary gland enlargement had been coincident with the tumor growth.

The physical examination showed a rather robust type of male lying quietly in bed. The left side of the face was swollen from what was apparently a superficial tumor mass. The left eyeball showed marked proptosis and conjunctivitis with increased lacrimation. There was some bloody discharge from the left side of the nose, and the nose was enlarged on the left.

The skull was of normal contour with some exostoses in the supraorbital portion of the left frontal region of the orbital foramen. The right pupil was somewhat irregular. It reacted slowly to light and accommodation. The right eye movements were normal. The left eyeball showed marked proptosis and the left pupil was smaller than the right. It reacted slowly to light and accommodation. The movements of the left eye were limited.

The right side of the nose was patent. There was no tumor. The left side of the nose was completely occluded by a fungating tumor mass which protruded well into the lumen. The external nose was distorted to the left by the tumor. There was no swelling or tenderness over the right sinuses, but there was on the left. The mouth was edentulous. The gums were in good condition. There was some bulging at the site of the former bicuspids and first molar above on the left. This area fluctuated slightly on pressure and was sensitive to palpation. The tongue was clean and protruded in the midline. There was a small tumor, about 2 or 3 cm. in size, projecting into the nasopharynx which could be seen with the postnasal mirror. The pharynx was slightly injected. The breath was foul. The tonsils were small. There was a slight anterior cervical lymph adenopathy palpable on the left, but not on the right. The submaxillary gland on the left was 4 or 5 cm. in size and sensitive to palpation.

The clinical diagnosis was probable malignant tumor of the left face, probably originating from the left ethmoidal cells and orbit.

On December 30, 1933, tissue was removed from the tumor mass protruding beneath the left inferior turbinate, and sent to the laboratory.

On December 31, 1933, under local anesthesia, a small cervical gland was exposed and a biopsy taken for metastasis.

On January 6, 1934, under ether anesthesia, a lateral rhinotomy, through the left cheek, was performed. A tumor mass was found to have broken through the anterior maxillary wall. The left maxillary sinus, ethmoid, and part of the sphenoid were com-

pletely filled with this tumor mass. The floor of the maxillary sinus was necrotic, but had not broken through the oral mucous membrane. All of the median wall of the orbit was destroyed by tumor tissue. Invasion of the pterygomaxillary fossa was evident. The nasoantral wall and all of the turbinates were removed and the tumor tissue in the maxillary sinus, ethmoidal cells and sphenoidal sinus exenterated. The cheek wound was left open for the insertion of radium.

On January 5, 1934, the radiologist reported enlargement of the heart to the left combined with some widening of the aortic arch. There was marked fibrosis in the hili and bronchial system. There was no evidence of pulmonary tumor metastases or of pleural effusion.

On December 27, 1933, the urinalysis was negative. On the 28th the erythrocytes were 4,390,000 and the leukocytes 7,850. The blood Wassermann and Eagle tests were negative on December 27, 1933.

The final diagnosis was rhabdomyoma of the left maxillary sinus, nose and orbit which probably originated from the internal pterygoid muscle. Imperatori[40] has reported a case of rhabdomyoma of the larynx.

From Jan. 1 to 6, 1934, the patient received four treatments with deep roentgen-rays.

On January 10, 1934, 50 mg. of radium, protected by 2 mm. of lead and 1 mm. of brass, was implanted ½ inch below the skin in the left maxillary sinus through the surgical incision. This was left in place 36 hours and reinserted in the ethmoid region for 24 hours. The eye was protected by an additional lead strip. The left maxillary sinus was washed a few times with saline or boric acid subsequent to the operation and before his discharge. His progress while in the hospital was quite satisfactory. He was discharged to the outpatient department as improved on January 24, 1934. He received additional radiation subsequently. He did not do very well, however, and died in another hospital on May 10, 1934.

Tissue specimens were removed from the nose and antrum, submaxillary gland and later from the antrum, sphenoidal and ethmoidal sinuses. All tumor tissue was firm, reddish externally, with solid yellowish-white cut surfaces. Microscopically, an extremely bizarre growth was seen with essentially similar appearance in tissue from all sites. The tumor cells were large oval or elongated, with abundant, often vacuolated acidophilic cytoplasm and angu-

lar reticular nuclei with prominent nucleoli. In an occasional cell cross striation resembling that of skeletal muscle was plainly seen. Mitoses were rare. The tumor cells formed small, solid groups, separated by a network of connective tissue fibrils. The surface in many sections was covered by pseudostratified columnar epithelium. Extensive zones of hemorrhage and necrosis were present, with varying numbers of infiltrating leukocytes. Spicules of bone were present in some sections. Tissue from the submaxillary gland was almost completely necrotic, but a small zone containing tumor cells was found.

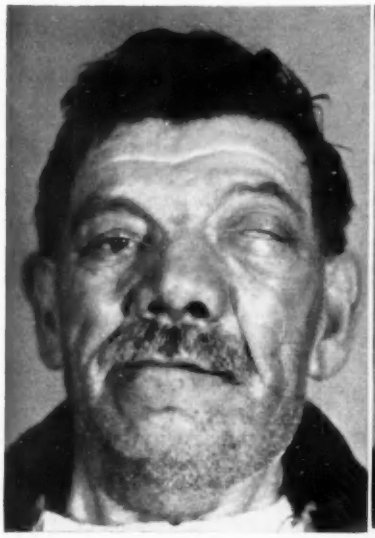

FIG. 16—(Spencer and Black)—Case 11. Photograph of patient taken a few days after admission with Rhabdomyoma of left maxillary, ethmoid, sphenoid, orbit and nasopharynx.

FIG. 17—(Spencer and Black)—Case 11. Photograph taken a few days after radical operation for Rhabdomyoma of left maxillary, ethmoid, sphenoid, orbit and nasopharynx.

The tumor was a malignant rhabdomyoma, embryonal in type, but lacking other tissue elements which might be considered teratoid. For the most part the tumor cells lacked cross striations, but were acidophilic, and were similar to those in which cross striation of the cytoplasm was distinct.

The fact that the submaxillary gland was involved might suggest an origin as a mixed tumor, but as no evidence of teratoid elements was found in any location, it seems more reasonable to conclude that the primary site was antral.

Such tumors are of rare occurrence, most often in association with other constituents of mixed tumors. The genito-urinary tract is the most frequent site. Other locations include the heart, testis, lung, breast, tongue, stomach and parotid.

The malignancy of rhabdomyoma is variable. Some of slow growing encapsulated tumors, while others are highly malignant. This tumor was invasive, nonencapsulated, and appeared in all respects malignant.

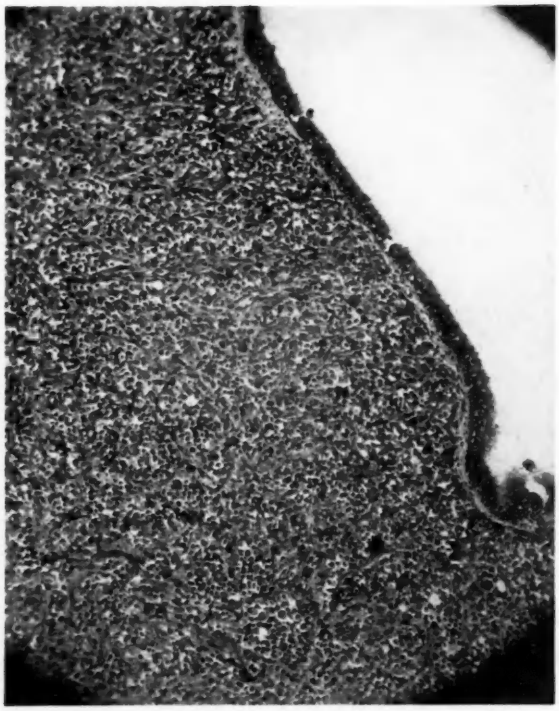

FIG. 18 — (Spencer and Black) — Case 11. Rhabdomyoma of left maxillary sinus, ethmoid, sphenoid, orbit and nasopharynx magnified 75×.

PATHOLOGY. — The type of tumor in three of the cases is not known, as tissue for examination was not available, and biopsy reports are not on record. In the other eight cases a remarkably wide variety of tumors was found, including three unusual tumors.

SUMMARY OF HISTOPATHOLOGY. — A series of eleven cases of tumors involving the nasopharynx and accessory sinuses included

three tumors of undetermined type (no biopsy), one transitional cell carcinoma, four squamous cell carcinomata, one neurosarcoma, one plasmacytoma, and one rhabdomyoma. The histological characteristics of these tumors are described.

RESULTS. — The "follow-up" shows that only two are living. One of the two has almost survived the five year period and the other has been under observation two years since his operation. Four are known to be dead. One of these was very advanced and inoperable at the time of admission. We have not been able to trace five of the patients.

Cooper[37] previously reported in 1934 the cases of plasmacytoma and rhabdomyoma included in this series of eleven cases. With his permission the two cases are included.

SUMMARY AND CONCLUSIONS. — Earlier diagnosis, especially for intelligent private patients, early removal of the tumor by surgical diathermy, preceded on the operating table by a biopsy, the use of the cautery, radium and the roentgen-ray have all helped to save human lives. Surgical removal of the superior maxilla is now rarely used. The death rate from this procedure, in the hands of our best otolaryngologists, has been quite high. Subsequent observation for any recurrence, with proper treatment for the growth, has also improved the results.

This group of eleven cases shows the variation in malignant tumors in a relatively small series. It also shows the necessity of a biopsy before any type of treatment is started, except in very late inoperable cases. The surgical, roentgen-ray and radium treatment, cauterization, and surgical diathermy had to be used according to the type of tumor, as shown by the biopsy, and the extent of sinus as well as orbital involvement.

BIBLIOGRAPHY

1. Houser, Karl M.: *Diagnosis and Treatment of Primary Malignant Neoplasms of the Maxillary Sinus.* Arch. Otolaryng., 18:643-650, 1933.
2. Lederer, Francis L.: *Problems Involved in Diagnosis and Treatment of Malignancies of Nasal Accessory Sinuses.* Wis. Med. Jour., 36:263, 1937.
3. Phillips, Wendell C.: *Primary Epithelioma of the Antrum of Highmore.* Jour. Laryng., Rhin. and Otol., 13:325, 1898.
4. New, Gordon B.: *Malignant Tumors of the Antrum of Highmore; End-Results of Treatments.* Arch. of Otolaryng., 4:201-214, 1926.
5. Spencer, Frank R.: *Carcinoma of the Tongue with a Review of Fourteen Cases.* Trans. Amer. Laryng., Rhin. and Otol. Soc., 42:95-111, 1936.

6. Davison, Charles, and Kahr, Sidney: *Carcinoma of the Sphenoid Sinuses with Invasion of the Cranial Cavity.* Arch. Otolaryng., 14:16-22, 1931.
7. Myerson, M. C.: *Carcinoma of the Orbit and Ethmoid Removed by Surgical Diathermy.* Laryngoscope, 39:185-186, 1929.
8. Fenton, Ralph A.: *Some Annoyances in the Management of Malignancies of the Accessory Sinuses.* Ann. Otol., Rhin. and Laryng., 43:39-46, 1934.
9. Schall, LeRoy A.: *Exophthalmos Complicating Irradiation.* Trans. Section on Lar., Otol. and Rhin., A.M.A., 1937, in press.
10. Hill, Frederick T.: *Malignant Disease of the Sinuses and Nasopharynx, in the Small Hospital.* Ann. Otol., Rhin. and Laryng., 46:158-168, 1937.
11. Bryan, Joseph H.: *Report of a Case of Papillary Carcinoma of the Maxillary Sinus.* Ann. Otol., Rhin. and Laryng., 41:983-989, 1932.
12. Stone, C. E.: *Primary Carcinoma of Maxillary Antrum.* Jour. Ind. State Med. Assn., 23:315-316, July 1930.
13. Hartz, William: *Intranasal Malignant Growths.* Laryngoscope, 45:844-853, 1935.
14. Beck, Joseph C., and Guttman, M. Reese: *Carcinoma of the Tongue with Special Reference to Treatment by Irradiation.* Ann. Otol., Rhin. and Laryng., 41:924-932, 1932.
15. Beck, Joseph C., and Guttman, M. Reese: *Malignant Disease of the Nose and Nasal Accessory Sinuses.* Ann. Otol., Rhin. and Laryng., 41: 1-20, 1932.
16. Quick, D.: *Treatment of Malignant Growths of the Nasal Accessory Sinuses and Nasopharynx.* Radiology, 14:191, 1930.
17. Barnes, Harry A.: *Malignant Tumors of the Nasal Sinuses.* Arch. Otolaryng., 6:123, 1927.
18. Woodman, E. M.: *Discussion on Treatment of Malignant Growths.* Proc. Roy. Soc. Med., Part 1-2, Sect. on Laryng., 15:35-38, 1921-1922.
19. Davis, E. D. D.: *Malignant Growths of the Upper Jaw and Antrum.* Lancet, 2:1090-1092, 1920.
20. Thompson, Geo. H.: *Malignant Neoplasms of the Antrum.* Ann. Otol., Rhin. and Laryng., 36:715-732, 1927.
21. Nash, C. Stewart: *The Management of Malignancies of the Sinuses.* Ann. Otol., Rhin. and Laryng., 44:220-225, 1935.
22. Shutes, Milton H.: *Cured Carcinoma of Nose and Antrum.* Laryngoscope, 44:924-925, 1934.
23. Greene, D. Crosby: *Carcinoma of the Antrum.* Amer. Jour. Roentgenology, 9:551-599, 1922.
24. Barnes, Harry A.: *The Combined Operative and Radium Treatment of Malignant Disease of Nasal Accessory Sinuses.* Boston Med. and Surg. Jour., 183:648-656, 1920.
25. Öhngren, L. G.: *Malignant Tumors of the Maxillo-Ethmoidal Region: Clinical Study with Special Reference to Treatment with Electro-Surgery and Irradiation.* Acta Otolaryng. Supp., 19:1-476, 1933.
26. Johnson, F. M.: *Radium Treatment of Carcinoma of the Antrum.* Surg., Gynec. and Obst., 38:819-822, 1924.
27. Robinson, G. Allen: *Radium in Malignant Tumors of the Nasal Sinuses.* Laryngoscope, 43:285-286, 1933.
28. McCoy, John: *Sarcoma of the Antrum, Ethmoid and Sphenoid. Treated by Radium. Case Report.* Trans. Amer. Laryng., Rhin. and Otol. Soc., 38:308, 1932.
29. Spencer, Frank R.: *Primary Cholesteatoma of the Sinuses and Orbit. Report of a Case of Many Years' Duration Followed by Carcinoma and Death.* Trans. Amer. Laryng., Rhin. and Otol. Soc., 36:543-549, 1930.

30. Spencer, Frank R.: *Chronic Granulomata of the Nose and Nasal Accessory Sinuses. The Nose, Throat and Ear and Their Diseases.* Edited by Jackson and Coates. W. B. Saunders Co., Phila., 1929, pp. 186-197.
31. Harmer, Douglas: *Discussion on Treatment of Malignant Growths.* Proc. Roy. Soc. Med., Part 1-2, Sect. on Laryng., 15:40-41, 1921-1922.
32. New, Gordon B.: *Squamous Cell Epithelioma without Recurrence After Ten Years.* Surg. Clin. of N. Amer., Aug. 1933, 13:871-873.
33. New, Gordon B., and Cabot, Clyde M.: *The Curability of Malignant Tumors of Upper Jaw and Antrum.* Trans. Amer. Laryng., Rhin. and Otol. Soc., 41:584-590, 1935.
34. Ewing, James: *Neoplastic Disease.* W. B. Saunders Co., Phila., 3rd Ed., pp. 875-877.
35. Stewart, F. W., and Copeland, M. M.: *Neurogenic Sarcoma.* Amer. Jour. Cancer, 15:1235-1318, 1931.
36. Walgren, A.: Virchow's Arch. f. path. Anat., 232:381, 1921.
37. Cooper, Kemp G.: *Plasmacytoma and Rhabdomyoma of the Paranasal Sinuses.* Arch. Otolaryngol., 20:329-339, 1934.
38. Blumenfeld, Louis: *Plasma Cell Tumors of the Nose and Nasopharynx.* Ann. Otol., Rhin. and Laryng., 45:436-446, 1936.
39. Jackson, H.; Parker, F., Jr., and Bethea, J. M.: *Studies on the Diseases of Lymphoid and Myeloid Tissue: II. Plasmacytomata and Their Relations to Multiple Myelomata.* Amer. Jour. Med. Sc., 181:169, 1931.
40. Imperatori, Charles J.: *Rhabdomyoma of the Larynx. Report of a Case.* Trans. Amer. Laryng. Ass'n, 55:139.

DISCUSSION

Dr. Harris P. Mosher, Boston: Knowing the writer of this paper as I do, his paper is what I fully expected it would be. It is one of the most thorough and systematic papers I have ever read, beautifully presented.

Permit me to make a wisecrack, so that you will recognize me, namely, if the paper is published as sent to me, the Transactions will be double their usual size — sixty-seven pages, seven of bibliography. The histories of the cases are most meticulously given. I am not joking when I say they could be used as models for medical students and for many of the rest of us. I thought his introduction was especially striking.

As the writer pointed out, in his series of eleven cases, three were very unusual. Not only were the histories of the cases given most fully, but the pathological reports were beautifully clear and complete. These could serve as models in their line.

It is dangerous to disagree with New, but I do not see why his biopsy should be taken on the operating table instead of a few days to a week before. It is not a bad plan, in my opinion, when you can, to plan your operation beforehand. The use of the cautery has never appealed to me. I thought that the full removal of the superior maxillæ had been given up long ago in favor of fractional removal. I used it only once a few years ago. Fractional removal is not followed by any appreciable operative mortality.

The full Moure operation gives the fullest approach and the quickest. I have been accustomed to leave an opening in the cheek for observation and the subsequent implantation of radium and to watch for recurrence.

This opening proved very hard to close. Schall has convinced me that leaving the opening under the upper lip is equally practical. It has the advantage that the patient is not handicapped or ostracized by any external deformity.

As I see it, the big point in the management of these cases is whether to use surgery supplemented by radium and x-ray or to use radium and x-ray alone. We face the same problem in regard to cancer of the larynx. The x-ray people — notably, Quick — are becoming more and more positive, if possible, that surgery should give way to x-ray and radium. His figures are very impressive. If three or four equally well trained men can duplicate his results, more of us who depend primarily on surgery will have to sit up and take notice.

I cannot help returning to the case records in closing. As I said before, I have never read such thorough preparation. Now and then I picked up a new bit of description, for example, "There was no pulse deficit." This is striking: "He chewed before he took sick." "He had no trouble starting or stopping the stream." Could you ask for anything more? I couldn't.

Dr. Gordon B. New, Rochester, Minn.: It seems to me that Dr. Spencer has done a wonderful piece of work in reviewing our present knowledge of the method of taking care of these cases. I do not know of any particular area of the body in which there has been such a marked improvement in the treatment of malignant tumors as there has been in the malignant tumors of the upper jaw and the antrum in the last twenty years. We all know of Öhngren's work in Stockholm and what wonderful results he obtained in the care of these cases. Most of us remember, too, that when these patients came to us twenty years ago, the whole resection of the jaw was done. Since that time there has been considerable evolution in the treatment of these cases — that is, I personally used the old soldering iron with water-cooled retractors protecting the cheeks and tongue. Then we developed the large electric cauteries, and then we added to that radium and x-ray, then the use of surgical diathermy, until now I believe the combination of these methods of treatment has really added considerable to the outlook in these cases.

It was brought out by Dr. Spencer that early diagnosis, of course, is important, and I feel, too, that the microscopic examination on the operating table is important. Within the last month I have seen two cases where x-ray treatment was used outside of the face without a microscopic diagnosis, thinking that these tumors were malignant. One was a huge giant cell tumor and the other was a large fibroma. Biopsies were made at the time and then treatment accordingly given. These benign tumors, of course, were removed surgically in the usual manner. These cases I prefer to take care of with intratracheal anesthesia, and then in general an exploration is made above the alveolar process. In about 10 per cent of the cases, the patient loses the eye on the involved side, and, of course, in many there is a large perforation in the mouth where later a dental plate is used.

In these cases I do not believe a pedicle flap brought up from the neck to close these openings is advisable, because in those cases I have never seen where it was possible to put a dental plate on top of it. The large openings in the cheek that are left are later closed, and I feel, too, they should be left open at least a year until we are sure that the patient is well.

Back in 1920 I reported 33 of these cases, bringing out the fact that the end results had fewer recurrences following the use of the soldering iron and radium than there were with the whole resection of the jaw. Two years ago I reported 295 tumors of the upper jaw and antrum. These included, of course, all tumors of the upper jaw and antrum that were operated on previous to 1929, so that all of these cases had a chance to live five years. These included, of course, the low grade tumors, the adenoma carcinoma, tumors of that type that are a very low grade type of malignancy. Of this entire group we had 91 which were apparently primary antrum tumors. Of course, it is sometimes difficult to determine a primary antrum tumor from a secondary one, but apparently there were 91 operated on previous to 1929.

I was able to trace in five years 75 of these patients, and of those we had 40 per cent who were alive at the end of five years without recurrences. At the end of ten years we were able to trace, as I recall, 49 of these patients of whom 24.5 per cent were alive and without recurrence. This is with the combination of the use of surgical diathermy, radiation, the cautery, and x-ray.

Of the entire group of 295 patients who were operated on with this type of tumor, in five years I was able to trace 237 by letter or through their doctors, or I had some definite means of knowing whether they were alive or dead, and that is really what we wanted to know. This includes the low grade tumors as well as the highly malignant, the ones of the alveolar process, which are practically all grade 2, low grade tumors. Of these there were 54 per cent that were well five years later. At the end of ten years we were able to trace 151, and of those there were 35 per cent that were well ten years later.

I think a great deal of this result of these cases is due to the work of Öhngren of Stockholm, who has been a great advocate of this type of treatment.

Dr. Joseph C. Beck, Chicago: To follow Dr. Spencer's presentation and the two discussers, I think we might say there is really nothing to be added, but I do want to read a note that I wrote shortly after I received Dr. Spencer's paper, and I will finish what I have to say about his paper directly.

"I received your draft of your paper on malignant diseases of the nasal accessory sinuses and have read only part, but will be ready to discuss it. You leave very little to a discusser, for the subject is very well covered in your communication."

Now this paper, as Dr. Mosher has said, ought to be published in toto in order to get the essentials of a man who makes his report on a fairly small number of cases, to be sure, but it is the way they were observed and written, and that is what most of you are interested in, because you rarely have the opportunity such as Dr. New has. I have been active in this work for a number of years, and when I hear these reports I just have to shut up, that is all. The number is simply impossible for any other man to compare with. There is such a wonderful group. So it is very refreshing sometimes to hear a report such as Dr. Spencer has given.

Dr. New referred to Öhngren's work, who is an associate of Professor Holmgren. For six weeks I had the real pleasure, indeed, of visiting in Stockholm, in the Sabbatsberg Clinic, and I watched these cases, not as they operated them, but in the review over that period, in this hospital under the care of this gentleman. In Sweden, all cases of malignant disease are sent to Stockholm, both for the surgery as well as the radium activities and treatment, and there you can observe the management of cases such as these. Plastic surgery is his great field, although he has the complete subject at hand.

There is one point that Dr. New discussed in Dr. Spencer's work and presentation and Dr. Mosher referred to it, regarding the other sinuses, particularly Schall's recent report on the orbit. When we discuss malignant diseases of the sinuses, it is quite a different story from that of "malignant diseases of the antrum" and "malignant diseases of the spheno-ethmoid region," where we have the orbit as well as the intracranial cavity to consider, because patients do not die, as a rule, from the malignant disease, but they die of meningitis from the extension of the process causing infection. That has been the greatest difficulty.

Something has been said about different types of surgery versus the irradiation; also reference has been made to the great enthusiasm of the radium and x-ray experts. They must have enthusiasm. Enthusiasm is their great field right now. We must be very fair and observe the cases that are treated. In a very large institution, probably the largest in this country, where veterans are sent in large numbers, the Hines Veterans' Hospital, I have had a chance to observe as consultant in that service both these treatments over quite a period of time. These cases are increasing, as you know. These people are getting old, and there is a larger number of cases.

I will say to you, you have to go some yet to surpass the cases treated with surgery combined with the irradiation compared to cases that are treated with the irradiation alone. It is very important that we stick to our game of removing the growth as far as we can. Naturally, we should not endanger lives of patients by radical operation on the spheno-ethmoid region and kill them by meningitis, but wherever we can thoroughly remove the growth and follow with treatment, according to methods of the physicist and the radium x-ray therapist, I think we will have better results. I can see in long periods of cures that the results in treating this disease have been steadily increasing. I think the prognosis, especially in the adult type of carcinoma (and that is what they mostly are), or the chances for saving the lives of these patients, is really very good.

DR. FRANK R. SPENCER, closing: We have used Moure's operation some, but one of our younger men, Dr. Cooper, had his training under Dr. Mosher, and he has taken a fancy to this external rhinotomy; that is why I was able to show it to you in some of the photographs of patients. We quoted Öhngren's work in our paper. We were led to do that by seeing a quotation from it in Dr. New's article. We have taken it from Dr. New's article and given Dr. New credit.

Dr. Beck speaks of this being an interesting series. When I looked over the cases of rhabdomyoma, plasmacytoma, and neurogenic sarcoma, multiple in different parts of the body chiefly in the left maxillary sinus, I felt they might be of interest to you. Certainly they were of interest to those of us who took care of the patients.

I hope those of you who did not hear Dr. Schall's paper at the last A.M.A. meeting will take time to read it. It deals with one very important symptom which we often encounter, and that is exophthalmos. If the eye has not been lost by malignant disease of the orbit, it may be lost, and several of these patients lost an eye.

Dr. Beck spoke of the importance of the radiologist's work. We all certainly need the radiologist when we try to treat malignant disease, and a few patients of this series, who are still living, owe their lives as much to what the radiologist did as to anything we did.

I appreciate the discussion by three such loyal friends of many years as Dr. Mosher, Dr. New and Dr. Beck.

PLASTIC REPAIR OF DEFORMITIES ABOUT THE LOWER PART OF THE NOSE RESULTING FROM LOSS OF TISSUE

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INTRODUCTION. — Loss of tissue in the lower part of the nose, either from disease or injury, causes, in the aggregate, a large number and variety of deformities. The origin and etiology of the defects are usually of secondary importance in their management, but the cosmetic and functional anomalies present problems of great importance.

The extent of the deformity provides only a fair basis for classification. It is difficult to arrive, however, at any other systematic approach, and if the following grouping of cases has any value, its only basis of reason lies in its serving as a clinically practical means of subdividing these deformities. In my own experience it seemed to fit into the usual methods of treatment better than any other grouping. Therefore, they are classified below as follows:

(1) Loss of the skin;
(2) Loss of the alæ;
(3) Loss of the tip and alæ;
(4) Loss of the inner lining.

ETIOLOGY. — These deformities may be caused by automobile accidents or other civilian injuries, resulting in unsightly scars or discoloration of the skin from minute particles of dirt; by pigmented nævi or cavernous angiomas; by superficial basal cell carcinoma, and finally by chronic ulcerative lesions caused by tuberculosis and syphilis.

Deformities resulting from the loss of skin present lesser difficulties than those involving the borders of the nostrils and the inner lining of the nose. In the former case, the problem centers about the most feasible method of supplying skin covering, while in the latter group of cases we are dealing with more complex

problems; that is, reforming the inner lining as well as the skin and the establishment of the contour of the tip and borders of the nares within the normal limits. To my mind the most difficult part of rhinoplastic surgery centers around the tip of the nostrils.

GROUP I. — *Deformities from Loss of Skin Only.* (Cases I, II, III)

The first step is the excision of all pathological or abnormal tissue. After excision, two methods of repair are available, and the choice between them depends upon the extent of the original deformity. We may use, first, a full thickness skin graft, or second, a pedicled graft.

If the lesion is superficial, such as in scars, pigmented nævi or angiomas, preference is given to the full thickness skin graft. Transplants are taken either from the back of the ear or from a covered part of the body. Experience has shown that the skin of the ear is superior to the other parts, as it blends more harmoniously with the surrounding skin, producing a good cosmetic result.

Preference is given to full thickness grafts rather than razor grafts. Although razor grafts have a greater chance to take, yet there seem to be serious objections: (1) they do not provide enough thickness to harmonize with the contour of the surrounding tissues; (2) as the grafts are taken from the distant covered parts of the body, such as the arm and leg, both the texture and the color of the transplants are more objectionable.

TECHNIQUE. — An oval-shaped area of skin is excised from the back of the ear. Any excess fat is trimmed off. Then suture the transplant accurately to the borders of the defect after stopping all the active bleeding. Cover the transplant with crepe de lis or any other non-adhesive dressing supplementing with sufficient surgical dressing to absorb serum. Dress with a pressure bandage having in mind that immobilization of the grafted part is one of the most important steps in success. I think the most serious period is the first twenty-four hours, although one rarely disturbs the dressing for four to five days. Not infrequently, I have opened the dressing after twenty-four hours without causing injuries to the graft.

Pedicled flaps are chosen whenever more skin is needed than can be borrowed from the ear, or whenever the nature of the defect requires fat as well as skin for its repair. Such defects more commonly result from malignant disease.

The donor areas of pedicle grafts are, in order of preference: (1) the forehead; (2) the arm; (3) other parts, such as the abdominal wall, the chest, or neck.

The forehead as the donor area should have the first consideration for several reasons:

(1) The skin of the forehead harmonizes with that of the nose more completely than that of any other part and has good cosmetic value.

(2) It involves fewer steps in operative procedure than other methods. It it not necessary to repair the defect in more than two stages.

The first stage consists of removing the flap from its bed and suturing it over the defect. The second stage is to transfer back the unused part of the flap to the forehead and into the defect of the forehead. The remaining part of the forehead wound is closed either by approximating or by transplanting skin taken from some other part.

The obvious disadvantage lies in the resulting scar lines upon the forehead which are likely to become quite conspicuous under certain conditions. However, this deformity of the forehead is less conspicuous in women since they are able to cover the forehead with hair. Moreover, in the majority of the defects under discussion, it is possible to have enough surrounding skin tissue to repair the secondary defect of the forehead without resorting to skin transplantation; in other words, by direct closure of the forehead wound.

Patients with low foreheads and a profuse crop of hair are unfavorable for forehead grafts and it is necessary to use other methods. Therefore, it is fortunate that other methods, such as tube flap from other parts of the body, are available.

In suitable cases, especially in elderly patients where the skin is more or less loose, one can safely borrow skin from the surrounding areas of the face without causing conspicuous additional scars. A very convenient method consists of taking a tongue-shaped pedicled flap from the side of the upper lip, following the naso-labial line. The resulting scar is often hardly visible. In favorable cases, where there is an over-sized nose, a corrective rhinoplastic operation may provide enough loose tissue to cover the defect.

GROUP II. — *Loss of the Alæ.* (Cases IV, V, VI, VII, VIII)

Deformities of the alæ of the nose are quite common. The loss of tissue may include the entire wing of the nose or may affect only part of it. The deformities especially caused by injury may show varying kinds of distortion and a stenosis of the particular naris.

The alar cartilage normally is closely adherent to the skin on one side and to the mucosa on the other side, and this anatomical intimacy adds to its surgical difficulties. The inner lining often needs restoration before an adequate contour can be given the nares. The nostril ideally must be thin, resilient, and must maintain a symmetrical contour with its fellow. Many types of operation have been designed for restoration of the ala or alar defects.

A study of the literature on this subject reveals the following general methods of procedure:

(1) The upper remaining part of the nose is brought down to form the alar rim (Denonvilier, Tillaux, Mutter, Von Hacker).

(2) Flaps from side of the face and lip are transposed to fill the defect (Sédillot, Nélaton, Preidlsberger, Bouisson).

(3) Flaps from the forehead (Nélaton and Ambrédanne), arm, and in recent years since the discovery of tube flaps by Gillies, this method with various modifications is used by many plastic surgeons.

(4) Finally, free transplants of the entire thickness of the border of the ear have been used (Dredel, Joseph, König).

Various modifications of the above mentioned methods and procedures have been devised which are undoubtedly ingenious. Frequently, however, the question of the inner lining in these alar restorations, which we know today to be as essential as the outer skin, is not discussed.

In selecting a method or methods, it is important to choose one which necessitates a minimal number of operative steps, which causes minimal donor site defects, and which is the simplest to execute technically.

The writer, for all practical purposes, limits his own approach in the great majority of alar defects to two methods.

The first method consists of utilizing a section of the entire thickness of the lower part of the septum.

The second method consists of using the lower border of the defect itself to form the new alar rim; in the meantime, enough

mucous membrane is resected from under the nasal bones to form the inner surface of the deformed area. The raw area above the alar rim is covered by free transplant of skin.

The septal method (Case IV) is especially suitable for deformities involving the loss of the greater part of the ala of the nose, not over one-half inch in width.

This operation is done in three stages at monthly intervals. It consists of making three sides of a rectangular incision through the entire thickness of the septum beginning from behind the columella down to the base of the septum. Then the incision extends inward a distance of about 15 mm. to a point where a vertical incision toward the upper border of the septum is made. This square septal flap, which receives its blood supply from above, is sutured to the borders of the deformed nostril and held in position by an improvised splint made of gutta percha or dental composition, which is made to fit accurately the median side of the septal flap. In the second stage, the upper border of the septal flap is separated from its base and sutured near the tip of the nose. In the third stage the exposed surface of the flap, which is the mucous membrane of the septum, is removed with a sharp knife and replaced by a skin graft.

The final result of such an operation has been quite satisfactory. The border of the reconstructed nostril has the right contour and thickness, and harmonizes nicely with the other nostril. However, in selecting this method, one must realize that the septal mucous membrane is a delicate tissue and should be handled carefully. Care should also be taken to preserve an adequate blood supply to the pedicle. When cutting the upper border of the flap in the second stage of the operation, a sufficient amount of septal cartilage should be left to support the bridge. The nose will require frequent changes of dressing following the operation, as at times the secretions of the nose are quite troublesome. The septal perforation created is no real drawback to this procedure. The method is not suitable for defects that involve a part of the tip as well as the ala of the nose.

The second method: Utilization of the border of the defect to form the alar rim, with judicious modifications, has possibilities (Cases V, VI, VII, VII). It is a simple procedure. The cosmetic effect is good in that it results in minimum incision lines and it is free from the dangers of causing stenosis.

If the defect is similar to that in Fig. 9, where there is considerable loss of tissue, the air passage is partly closed while the border of the deviated ala is pulled upward by dense scar tissue.

After excision of the scar tissue two swinging flaps from the upper and lower remaining parts of the nostril are brought together to form the lower border of the ala. In utilizing the borders of the defect, the incision for the flaps is made through the skin only. The mucous membrane of the inner wall of the nose, being continuous with the flaps, is then carefully separated from the overlying skin and also from part of the nasal bone. The undermined mucosa is brought down with the skin flaps of the ala, to supply a sufficient inner lining. The final step is the repair of the skin defect above the alar rim, which consists of immediate transplantation of a full thickness skin graft taken from the back of the ear.

In other instances, if the picture of the defect is similar to that in Fig. 10, where there is no stenosis but a clear-cut defect with smooth margins, a curved incision is made a few millimeters behind the margin of the defect, extending through the height of the nose and cutting through the skin only, the mucous membrane is carefully separated from the overlying skin and part of the nasal bone and a whole flap is brought down and further steps are carried out as outlined above.

Of course, each case has its peculiar problems in the execution of operative details, yet by following the above general principles, I have been able to restore defects of moderate size with minimum operative trauma.

GROUP III. — *Loss of the Tip and the Alar Rims.* (Cases IX, X)

In the third group, there is considerable loss of tissue, usually involving the tip of the nose with its cartilaginous framework and the alar rims. To repair such deformities one is often obliged to transpose a large amount of tissue from the distant parts of the body because, usually, insufficient skin is available in the immediate neighborhood of the defect. Free transplantation is obviously not indicated.

I have at various times utilized flaps of the forehead, the neck, the scapula region, the chest and the arm, but after careful analysis of my cases, forehead flaps, on the whole, have many points in their favor as compared with other types. (These points have been

cited above.) In these cases, in addition, it is often necessary to transplant bone or cartilage also in order to give more accurate contours to the tip.

GROUP IV. — *Loss of the Inner Lining.* (Case XI)

The complete collapse of the tip or the lower part of the nose is caused by ulceration and subsequent destruction of the inner lining of the nose and its cartilaginous support, although the skin covering may remain practically intact. This type of deformity is found in patients suffering from tertiary syphilis and fortunately it is becoming rare today.

The question of treatment leads us toward finding a procedure whereby one is able to restore the framework and the inner lining of the skin of the nose.

The earliest attempts to raise the collapsed part of the nose was made by dentists who projected mechanical appliances through a perforation of the palate to fit under the skin of the nose (Kingsley, 1880; Claude Martin, 1889). Later, during and since the World War, this method in combination with surgery was found very useful both in carrying in transplants of skin during reconstruction of the nose and in using it as a permanent support for the nose. The writer for many years has been an ardent advocate of the judicious use of prosthetic appliances as an aid to reconstructive surgery of the face. However, he appreciates certain drawbacks of this method insofar as the difficulty surgeons have in not being trained as dental technicians and their being dependent on specially trained dentists to make their appliances. This is not always satisfactory. Moreover, unless the prosthesis is constructed to fit accurately the soft tissues of the nose and mouth, it is a source of irritation, and finally, mechanical appliances besides requiring careful handling by the patients occasionally are subject to breakage and must be repaired.

It is logical, therefore, I think, that any deformity that can be corrected effectively and with equal ease by surgical methods alone should receive preference to combined mechanical and surgical methods.

The method for the repair of the collapsed nose of this type consists of supplying the inner lining of the nose by turning inward two flaps of skin from the right and left sides of the upper lip. An incision is made around the alæ margins (see dotted lines in Fig. 26) extending through the base of the columella. The skin

is freed from the fibrous adhesions and is dissected freely over the whole nose, including the region of the nasal bones. A flap is prepared on each side by a curved incision beginning from the side of the ala in a direction corresponding with the lines of the upper lip. These flaps are dissected and turned inward, and sutured in place to form a new lining for the nasal cavity. No attempt is made to suture the base of the wings of the nose at this stage. This is left free, as shown in the diagram. In a later operation, the lower parts of these flaps, which are forming the inner lining of the nose, are cut through. Some of the excess tissue is excised and the base of the alæ are sutured in position.

In treating such cases, I found that there is a sufficient amount of fatty tissue in the flaps to give a good contour to the tip of the nose making it unnecessary to transplant cartilage or bone.

In presenting these cases, it has not been my intention to lay down rigid rules to be followed in the repair of various types of nasal defects. Rather, I have preferred to discuss critically a large number of different methods of repair in the hope that this may suggest to you advantages and disadvantages in most of these methods.

FIG. 1A — (Kazanjian)

FIG. 1B — (Kazanjian)

CASES — *Group I.*

Case I. B.R. — This little girl, seven years of age, was bitten by a dog three weeks previous to my seeing her, May 15, 1934.

There was a triangular raw area over the right side of the nose near the tip. The repair was done on May 25, under ether anesthesia and consisted of cleaning the surface of the wound by the removal of granulations. A full thickness graft taken from the back of the right ear was transposed to the nose and the result was very satisfactory (Figs. 1A and 1B).

Case II. K.H. — This patient (Fig. 2A), a boy of four and one-half years of age, had a congenital birthmark on the tip of the nose measuring 3 mm. in diameter. As he was becoming sensitive concerning his appearance, the parents wished to have the birthmark removed. The operation was performed under avertin-ether

FIG. 2A — (Kazanjian)

FIG. 2B — (Kazanjian)

anesthesia and consisted of excision of the birthmark, including a good margin of normal tissue. A skin graft was removed from the back of the ear, cut to fit the raw area on the nose and sutured in place. Care was taken to approximate the borders accurately. An external pressure bandage was applied. It was necessary to inspect the graft the following day because the bandage had become loose and moist. The transplant was found to be deep red in color. Daily dressings were then applied by using gauze saturated with warm normal salt solution. At the end of the tenth day, all dressings were discarded (Fig. 2B).

Case III. F.P. — The patient (Fig. 3A) was a 19-year-old girl injured in an automobile accident. Aside from injuries to the lip, the tip of the nose showed a left alar defect and considerable tattooing and scarring.

FIG. 3A — (Kazanjian)

FIG. 3B — (Kazanjian)

At the first operation, surgery was limited to the lip. At the second operation the scarred skin over the nose was excised freely and a full thickness skin graft removed from the back of the right ear and sutured into position. In the last operation, a small flap taken from the left upper part of the columella was turned across the vestibule to equalize the two vestibules (Fig. 3B).

Case IV. B.G. — This patient, age 18, a worker in a psychopathic hospital, was bitten by an insane patient. The detached tissue, involving the greater part of the wing of the left nostril was immediately sutured into position by a physician at the hospital. Unfortunately, this failed to "take" and it was necessary to remove it five days later (Fig. 4).

When the nasal wound had healed the repair of the nose was accomplished by utilizing a septal flap. A rectangular incision was made through the septum beginning from behind the columella down to the base of the septum, a distance of about 15 mm., where

FIG. 4 — (Kazanjian)

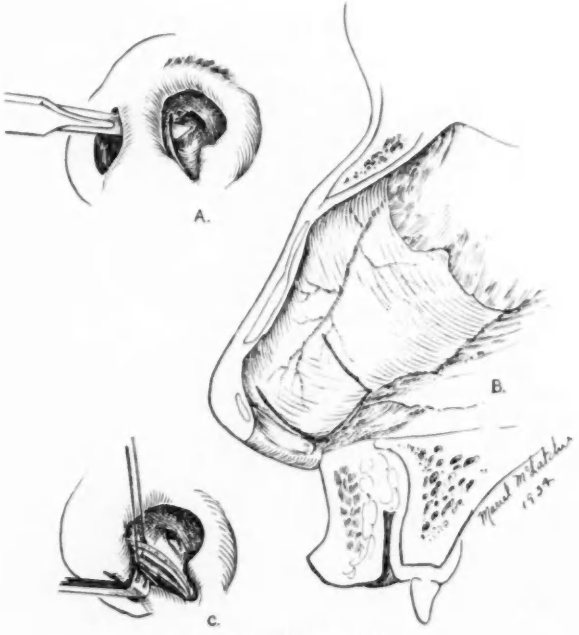

FIG. 5 — (Kazanjian)

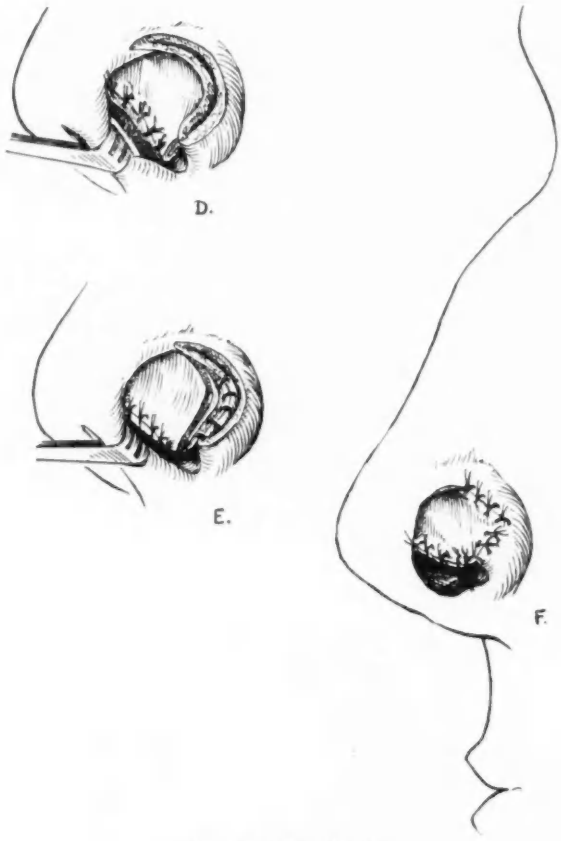

FIG. 6 — (Kazanjian)

a distal vertical incision to the upper border of the septum was made (Fig. 5). This septal flap, which received its blood supply from above, was sutured to the defective borders of the nostril (Fig. 6), and was held in position by a small gutta percha shield which was made to fit the nostril accurately. Three weeks later the upper end of the septal flap was severed from its base and sutured near the tip of the nose. At a later date the mucous membrane covering from the external surface was removed and replaced with a skin graft (Fig. 7).

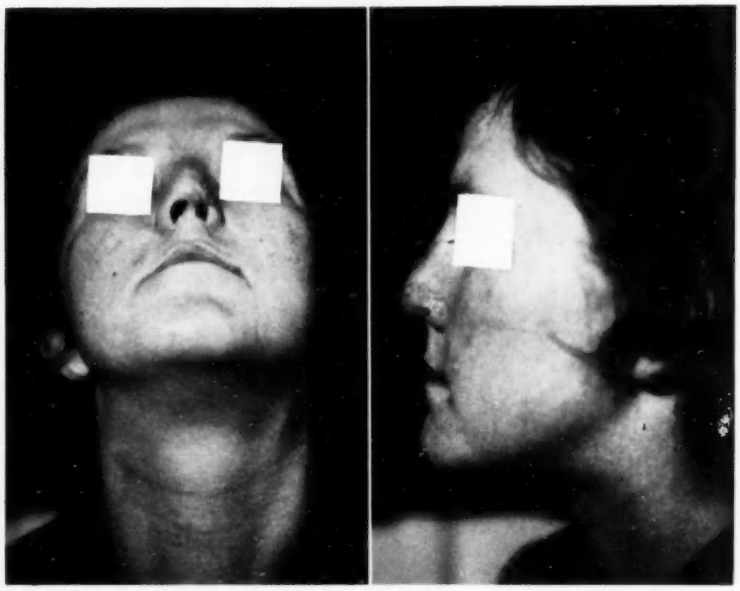

FIG. 7 — (Kazanjian)

FIG. 8 — (Kazanjian)

Case V. J.R. — This patient (Fig. 8), a boy of 9, was injured in an automobile accident on December 24, 1934, receiving lacerations about the face and nose. Examination on January 23 showed

considerable loss of tissue of the right lower border of the nose. The air passage was partly closed while the border of the deviated ala was pulled upward by dense scar tissue. On March 28, the patient was given an avertin-ether anesthesia. The scar of the center of the ala was excised. The line of incision of the lower part followed the curve of the wing while the one above was toward the tip of the nose (Fig. 9A). The mucous membrane lining of the inner wall was then freely undermined and cut through above the level of the nasal bones (Fig. 9B). Thus it was possible to bring down the distorted parts of the nostril to repair the defect (Fig. 9C). The triangular skin defect above was covered with a skin graft taken from the back of the ear (Fig. 9D). A mould made of dental

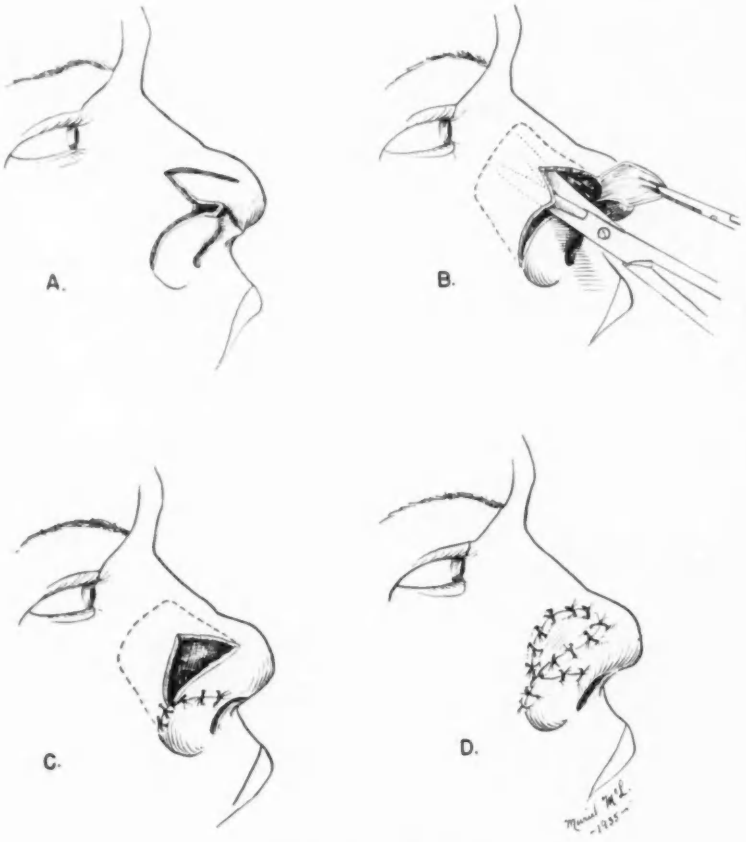

FIG. 9 — (Kazanjian)

FIG. 9E — (Kazanjian)

composition was fitted to the nostril and an external pressure bandage was applied. This was not touched for four days and then the first dressing was changed. The patient made a good recovery. The contour of the nose was good and there was no evidence of stenosis of the nostril. There was a slight irregularity near the tip of the nose which was to be corrected at a later date (Fig. 9E).

Case VI. W.H. — A colored man, age 32, was involved in a fight and he lost the right wing of the nose (Fig. 10). On examination, most of the right alar wing was missing and the healed borders of the wound were covered with scar tissue. It was explained that the repair of the deformity might involve several steps.

At the first operation the scar lines and scar tissue were resected and it was found that with the resulting gapping of the wound the lower border of the defect fell surprisingly near the proper border of the original nostril. The mucous membrane was incised high up under the nasal bone and with a slight amount of undermining it was possible to lower the border further. The oval gap remaining on the side of the nose was about 1.5 by 3.5 mm. in diameter. A full thickness graft of skin from behind the left ear was accurately sutured into the skin defect. The nose was packed and a pressure dressing applied (Fig. 11).

FIG. 10 — (Kazanjian)

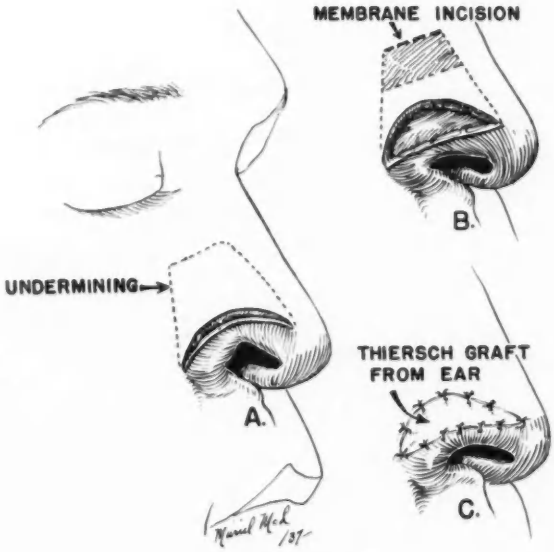

FIG. 11 — (Kazanjian)

FIG. 12 — (Kazanjian)

The nose was quite good in appearance after this first operation but the depression of the skin grafted area necessitated a second operation. At this operation an incision was made along the recent scar line near the edge of the nostril. The transplant and surrounding areas were undermined, exposing what was left of the original alar cartilage. From this cartilage, a sliver-like pedicle was freed and swung in such a fashion as to fill out the depression under the transplant. This pedicle was sutured in the new position and the wound closed with the usual nasal packing and external pressure dressing. This second step was so successful that no further surgery seems indicated in this case (Fig. 12).

Case VII. S.T. — This patient came to me with a history of radium treatments and one operative treatment of a "keloid" on the ala of the nose. Lately she had developed a persistent pimple in the same area. On examination I found a scarred and stenosed right naris with what seemed to be a recurrent basal cell carcinoma (Fig. 13).

At the first operation, Feb. 15, 1937, the malignant tissue with a margin of normal tissue was excised, leaving a further alar defect about 1 cm. in diameter. As the first step in repair, the scar tissue along the base of the nose was cut through and shaped to assume a position on the floor of the right nostril. A V-shaped raw space on the side of the septum was sutured together, which sim-

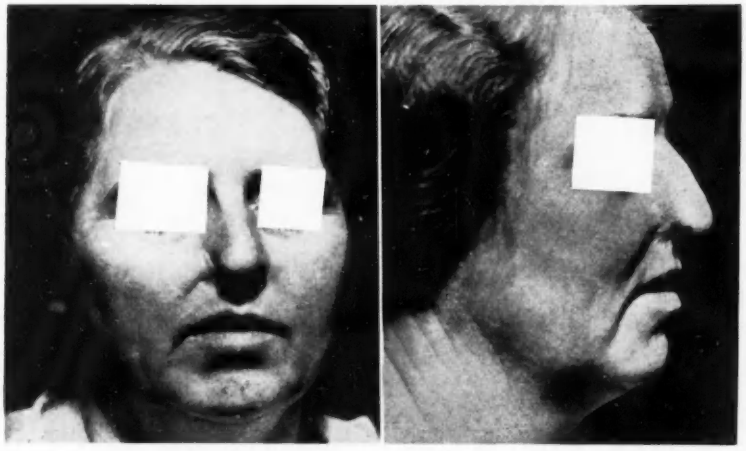

FIG. 13 — (Kazanjian)

plified the contour of the border of the defect. The ala was undermined up the right side of the nose to the lower edge of the nasal bones, separating mucous membrane and skin from each other. A full thickness graft was removed from the back of the ear measuring 2x¾ inches in diameter and divided into two portions. One part was sutured to the cleft along the lower border of the mucous membrane and the other along the skin border. The mucous membrane flap and the skin flap were pressed into the tunneled area,

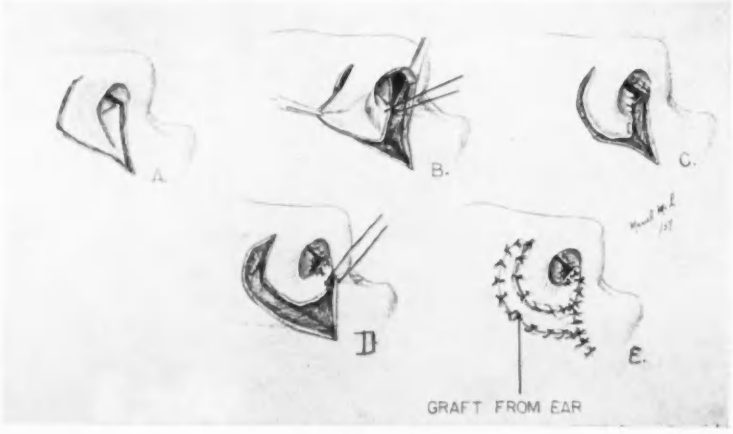

FIG. 14 — (Kazanjian)

thus creating a pouch between the skin and mucosa of the nose, and held in position with a dental composition splint supplemented by external pressure dressing. The purpose of this pouch was to provide in the future an epithelial lining to the inner side of the nose.

At the second operation on April 3, 1937, a diamond-shaped incision was made on the right side of the nose (Fig. 14). This freed the nostril and through the upper leg of the incision on the right side of the nose it was possible to pass the scalpel between the pouch and mucous membrane of the nose. This freed the pouch enough so that when the nostril was drawn down its epithelial lining became the new lining of part of the right nasal cavity. The new defect created externally was covered with a full thickness graft obtained from the postaural skin (Fig. 14E).

The results of this operation were quite satisfactory and it was decided that further surgery would be necessary only to thin the new alar wing. On May 26, 1937, an incision was made over the skin on the right side of the nose at the junction of skin and transplant. The skin over the wing of the nose was undermined freely and a considerable amount of underlying fibrous tissue was excised. A small ellipse of transplant was also excised. The borders were sutured together with kaldermic sutures.

The final results in this case were quite satisfactory. The nostril was of good shape and functionally adequate (Fig. 15).

FIG. 15 — (Kazanjian)

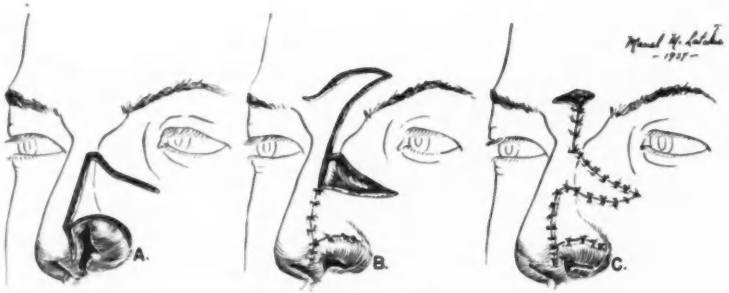

FIG. 16 — (Kazanjian)

Case VIII. J.C. — This patient was injured in an automobile accident on January 2, 1932. He suffered destruction of part of the left nostril near the tip of the nose (Fig. 16A). An examination on April 25 showed a definite loss of tissue with considerable cicatrization, forming a stenosis of the nasal passage on the left side. Operative treatment in this case consisted of (1) bringing the border of the retracted nostril forward by a through and through incision extending from the tip downward behind the base of the ala, thus forming the contour of the lower part; (2) the resulting defect was repaired by two sliding flaps, one from the side of the nose, the other from the forehead (Figs. 17 and 18).

FIG. 17 — (Kazanjian)

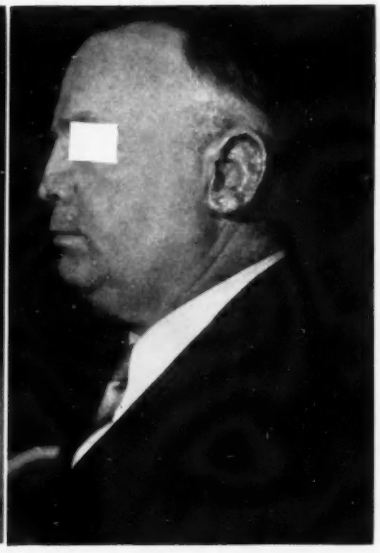

FIG. 18 — (Kazanjian)

FIG. 19 — (Kazanjian)

Case IX. J.H. — This patient was a worker in a psychopathic hospital, and was attacked by an insane patient who bit off the tip of his nose. As seen by the photograph (Fig. 19), the tip and part of the alæ are missing.

An elaborate plan of reconstruction was carried through in this case; we conceived the idea, not altogether new, of transposing part of the left ear, including the ear cartilage to the tip of the nose.

The first operation (Aug. 16, 1932) consisted of cutting an oval-shaped flap about one inch by one-half inch in size from the back of the ear, including the same amount of cartilage. This flap, with the ear cartilage, was turned in and imbedded under the skin of the back of the ear over the mastoid process (Fig. 20). In the second operation (Aug. 29, 1932), a long tube flap was made over the scalp extending from the median line to the back of the ear. The line of incision of the flap followed approximately the hair line. In the third operation (Sept. 27, 1932), the ear cartilage and the skin were separated from their base and transferred over the nose, the tube flap acting as a carrier (Fig. 21). In the fourth operation (Oct. 3, 1932), the tube flap was returned to its original position. The raw area of the ear was closed in the previous operation

FIG. 20 — (Kazanjian)

FIG. 21 — (Kazanjian)

by approximating the tissues; this worked well without causing undue distortion of the ear. In the fifth operation (Oct. 17, 1932), the surplus tissue over the tip of the nose was trimmed, which improved the contour (Fig. 22).

The final result of this operation was good; and the contour of the tip and alar rims looked quite normal. However, there are a good many objections to this method. (1) It was very difficult to keep the tube flap from becoming infected on account of the hair

FIG. 22 — (Kazanjian)

which grew rapidly over the carrier flap. (2) The great length of the tube flap constantly threatened the loss of the tissue that was actually needed for the repair of the nose. (3) It left a line of scar tissue at the hair-bearing area in back of the ear, which was quite conspicuous because of the absence of hair, but later disappeared. (4) Simpler methods for the same purpose are available, therefore, it is unnecessary to complicate the procedure.

The utilization of part of the ear is not a new procedure. According to Davis, Kredel used a triangle of cartilage from the ear; and König transplanted a section of full thickness of the margin of the ear into the defects in the ala. Joseph used a transplant of a full thickness graft. Later, Ivanissevich repaired the tip of the nose of a patient by taking a triangular piece of cartilage from the helix of the ear and using the thumb of one hand as a carrier.

My method differed in that the skin of the anterior part of the ear was not disturbed, and no deformity of the ear resulted. However, my great objection is not in the final result, which is good, but because of the complicated surgical procedure that was necessary to carry it through successfully.

FIG. 23 — (Kazanjian)

FIG. 24 — (Kazanjian)

Case X. H.N. — This thirty-three-year-old white woman had a history of lupus vulgaris of the nose of fifteen years' duration. This has been treated in several ways, apparently most effectively by x-ray. On examination, the tip of the nose was found to be destroyed and also part of the columella. The septum showed a large perforation. Scar tissue was present in the affected area, with no evidence of active lupus vulgaris (Fig. 23).

On June 21, 1933, a large skin flap was raised on the left side of the forehead with its base at the root of the nose. This was resutured in position to be utilized at the next operation as a delayed flap.

On July 8, 1933, the delayed flaps were freed again. The forehead flap was sutured to the denuded area on top of the nose. Half of the denuded area on the forehead was covered with a Thiersch graft.

At the third operation, Aug. 23, 1937, the pedicled flap was divided over the bridge of the nose and the upper part resutured to its original forehead site.

In three further steps or stages it was possible to repair the columella and to obtain a new nasal tip of fair contour. No further transplanting of skin was necessary (Fig. 24).

FIG. 25 — (Kazanjian)

Case XI. Mrs. D. — This patient reported on March 5, 1925, for plastic repair of the nose. She gave a history of a sore on the side of the nose which gradually developed extensive ulceration. Under specific anti-luetic treatment, the ulceration disappeared, but it left the nose in a deformed condition. Examination showed the lower part of the nose collapsed, mainly because of the destruction of the septum and the lower part of the alæ of the nose. The upper lip was retracted from the scars. The plan adopted for this case was to restore the inner lining of the nose and to repair the columella (Fig. 25).

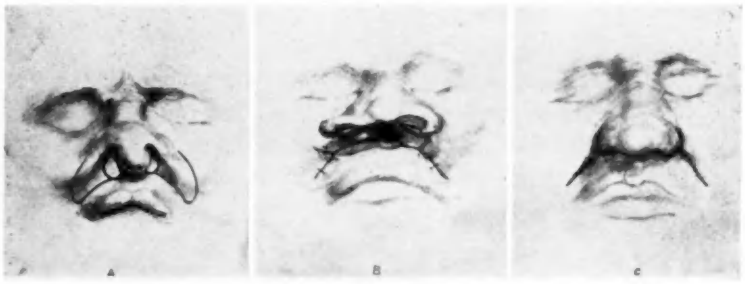

FIG. 26 — (Kazanjian)

FIG. 27 — (Kazanjian)

On March 21, 1925, the patient was operated on under general anesthesia. An incision was made along the side and base of the nose and the skin was freely separated from the underlying adhesions. Undermining was carried above the nasal bones. Tongue-shaped flaps were prepared from the side of the face following the naso-labial lines, and folded in to cover the raw surface under the skin of the nose (Fig. 26).

On April 4, 1925, the second operation was performed which consisted of cutting the base of the flap in order to suture the alæ of the nose in their proper positions. Eventually the columella was reformed by borrowing a section from the median line of the upper lip. In restoring the inner lining of the nose with flaps from the sides of the face, sufficient contour of the nose was obtained, making further operative work unnecessary (Fig. 27).

BIBLIOGRAPHY

1. Nélaton, Ch., and Ambrédanne, L.: *La "Rhinoplastique."* 1904, G. Steinheil, Paris.
2. Davis, J. S.: *"Plastic Surgery."* 1919. P. Blakiston's Son & Co., Philadelphia.
3. Martin, Claude: *"Prothese Immediate."* 1889. G. Masson, Paris.
4. Kingsley, Norman W.: *"Oral Deformities."* 1880. D. Appleton & Co., New York.
5. Sheehan, J. Eastman: *"Plastic Surgery of the Nose."* 1936. Paul B. Hoeber, Inc., pp. 93-103.
6. Smith, F.: *"Reconstructive Surgery of the Head and Neck."* Nelson's Loose-Leaf Surgery, p. 295.
7. Kazanjian, V. H.: *"Treatment of Nasal Deformities."* J.A.M.A., 84: 177-181, January 1925.
8. Blair, V. P., and Brown, B. J.: *"Nasal Abnormalities, Fancied and Real."* Surgery, Gynecology and Obstetrics, 53:797, 1931.

DISCUSSION

DR. F. A. FIGI, Rochester, Minn.: Dr. Kazanjian has presented a comprehensive review of deformities of the lower portion of the nose resulting from loss of tissue, together with methods for their repair, and he has shown us some very beautiful results.

Separation of these deformities into the several groups mentioned, depending on the situation and the extent of the tissue loss, is useful for didactic purposes and is helpful in planning repair. Actually, however, the loss is not often limited to the covering of the nose, its lining, or to the cartilages alone, but much more frequently involves at least two, if not all three, of these structures.

Procedures for the repair of these tissue losses are now fairly well standardized, although each case must of necessity be considered as an individual problem, taking into consideration the age, sex, and general condition of the patient, as well as the situation, extent, and etiology of the defect. In comparatively few of these cases is it possible to effect a repair — that is, satisfactory from both the functional and cosmetic standpoint by the use of full thickness skin grafts alone, because of the extent of the loss and the difficulty in maintaining asepsis and of applying adequate pressure over the lower portion of the nose. Accordingly, pedicled flaps, either with or without a lining, are usually required. The immediate problem then, in most of the cases, is to determine the area from which the necessary flap can be secured most satisfactorily. The area best suited will depend largely on the extent of the defect and whether it occurs alone or in association with other deformities of the face.

In the majority of the cases, the tissues of the forehead best meet the requirements because of the proximity, vascularity, color, and texture. In a male, however, because of the scar following the use of a forehead flap being unduly prominent, the procedure is rarely justified unless the individual's skin is unusually thin. The same objection holds for the use of flaps from the cheeks and from the nose itself, although tissues from these latter situations are not infrequently employed to advantage for economic reasons, since they can be transplanted more promptly and the reconstruction completed in a shorter period; also in elderly individuals.

(Nine slides were used to illustrate the discussion.)

Dr. C. R. Straatsma, New York: It is needless to say that it is difficult to discuss such a thoroughly prepared paper as this, and in so doing I wish to add two methods of nasal tip repair that have not been covered in this essay. The first is the use of the neck flap, and the second, the arm flap.

We all quite agree that when a flap is needed the material from the forehead is best, but in certain instances this is not available, either because of objections offered by the patient or there is not enough available tissue in this region due to previous trauma or disease.

The above methods of repair are fully discussed in the following articles: "Plastic Repair of Severe Radium Burns and Angioma." "Plastic Repair of Partial Losses of the Nasal Tip."

(Various slides were shown demonstrating methods of repair and results in cases in which both neck and arm flaps were utilized.

Dr. C. L. Straith (by invitation), Detroit: I know you have all appreciated this beautiful demonstration by Dr. Kazanjian. I know I have, and I don't want to bore you by going into any of the things that will really be repetition, and so I wish to suggest another method of repairing defects about the end of the nose that has not been mentioned.

The method was evolved to eliminate the forehead scars which follow the Indian method, and also to secure skin of better color and texture than

that available by the Italian arm pedicle method. I noticed that in most males, there is a hairless, thin, pliable area of skin of facial color and texture just beneath the ear, between the scalp and beard hairline. I decided to use this skin for nasal repairs.

The method has been used on a number patients, both men and women, four of whom will be demonstrated by means of slides. The method was first used on a 17-year-old boy whose nose had been severed by falling window glass when he was 2 years old. A tube pedicle was constructed on the side of the neck and terminated just beneath the ear. After two weeks, the pattern of the needed skin was outlined by incision and the skin undercut and resutured. One week later, this skin was transferred to the top of the nose on the tube pedicle. Seventeen days later, the pedicle was severed and the tissue shaped around the tip of the nose to form the tip, alæ and columella. The skin is practically hairless, of good color, and the resultant scar on the neck is much to be preferred to a forehead scar in men.

Left: For nasal losses in men, the skin is transferred from just below the ear. A tube pedicle flap is made over the right sternocleidomastoid muscle to the sternal notch and the transfer delayed for three weeks to establish circulation. *Center:* Scar tissue turned in for lining and skin beneath ear transferred to nose. Head case connected to chest holds head in proper position for seventeen days. *Right:* This skin makes an excellent repair. The color is good, skin is thin and easily shaped, practically hairless, and no scar is visible on the face. The neck scar is seldom noticed.

The other slides will illustrate the use of the method in the repair of several types of nasal defects. This method, then, seems to me to be particularly indicated for males where a forehead scar is objectionable and the use of other flaps would bring to the nose a skin of entirely different color and texture. This new method, therefore, has many advantages.

DR. V. H. KAZANJIAN, closing: I thank the essayists for their very kind words and discussions. Fortunately, in plastic surgery, there is more than one way of solving a definite problem, and you have seen pictures that have followed different routes with the same results. As I said before, in selecting the method of operation, we have to consider the question of hospitalization, the simplicity of our procedure, the locality and extent of the wound, etc.

Dr. Straith brought up the point that he has used the back of the ear. In fact, in one of my last pictures, where I showed the huge picture showing a lag flap taken from the scalp, the tissue was taken from the back of the ear and from the lobe of the ear and used quite successfully; yet I am inclined to think that this procedure requires far more hospitalization — and they have the hazards of infective processes setting in — than the simple method of using forehead flaps. I do not want to be dogmatic over it, but, at the same time, the forehead flap, I think, on the whole, has far more points in its favor than other types of flap I know of for the repair of large defects of the nose.

CLINICAL MANIFESTATIONS IN THE NOSE, THROAT, AND SINUSES IN DISEASES OF THE MONONUCLEOSIS TYPE

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WASHINGTON, D. C.

It is one of our members, Ralph A. Fenton,[1] who states that we have had the anatomic, rather than the physiological or pathological, approach to our studies in otolaryngology. This should give us pause and perhaps cause us to retrace our steps, so that we shall be enabled to consider, as did our medical forebears, the relationship of symptoms to physiology and to its diseased alteration, pathology. The studies in bone pathology by Mosher[2] and Furstenberg[3] throw light along the way. Along a similar line, the following views and observations, illustrated by cases, are presented on a rather obscure and unpredictable division of nose and throat pathology.

No one checking the clinical symptoms and blood pictures in otolaryngology can fail to be struck with the similarity in a group of diseases characterized by lesions of the ulcerative or membranous type in the nose, throat, and gums, associated with changes in the hemopoietic system. The borders of this group are nebulous, and it is a question whether the clinical manifestations presented in the fields of otolaryngology and stomatology are merely symptoms of an underlying blood dyscrasia, or whether nose, throat, mouth, and sinus infections in their varying degrees of severity, cause changes in the blood-forming organs, presenting a hemogram ranging from a slight leukocytosis in mild invasions, through a more serious invasion and less reparative effort, to a leukopenia; and with a still more severe infection, a mononucleosis. This mononucleosis is not to be confused with infectious mononucleosis, which appears to be an entity, and will be taken up later, but it is probably a phase of the next condition, possibly an agranulocytic syndrome in its transition stages. In the still more severe infections, with a grosser insult and inadequate defense, there would be an actual agranulocytosis. In this connection the influence of drugs

cannot be overlooked. Too many observers, especially Madison and Squier,[4] and Benjamin and Biederman,[5] have demonstrated the relationship between cause and effect, and have reproduced these syndromes by readministration of the barbiturates, amidopyrine, or others of the benzol series. This is probably an oversensitivity of the individual, if one considers the hundreds of thousands of doses either prescribed or taken over the counter, the proportion is very small. Direct proof (Witts[6]) of the culpability of amidopyrine has been obtained by observing the effect of a test dose on the white blood count of patients who have suffered from agranulocytosis. The number of white cells falls abruptly within a few hours, and at the same time the patient may complain of general symptoms such as malaise, sore throat, and pyrexia. Rigorous proof would demand the demonstration of sensitivity to amidopyrine in every case before the illness is accepted as due to it. On the other hand, T. T. Broughton,[7] of the University of Illinois, Department of Internal Medicine, states that the rubber factories in Akron have numerous cases of benzene poisoning, but agranulocytosis is rare. The deleterious effects of X-ray and radium come in this category, as well as the new panacea, the prontolin group. Kracke and Parker[8] suggest that the easily oxidizable benzene rings probably affect the marrow directly to produce the disease. Dameshek and Colmes[9] conclude that a "defective marrow may be of importance, as well as hypersensitivity and excessive dosages. Whether hypersensitivity is congenitally present or acquired during life will be subject to further investigation. No doubt, certain individuals, who are unusually sensitive to amidopyrine, develop a bone marrow reaction which results clinically in agranulocytosis." These drugs apparently act as bone marrow poisons, causing lack of maturation of the leukocytes, particularly the neutrophiles, and in addition the prontolin group affects the reds. Given a still more overwhelming invasion, there may be a complete abolition of reparative effort or a paralysis of the blood-reproducing system manifest in the blood picture as an aplastic anemia or a leukemia. The non-specificity of the causative agents is characteristic in these conditions. It is significant, however, that there is often a preceding history of severe upper respiratory infections in leukemias. The leukemias in this connection have been most thoroughly studied, and they are now considered in the group, leukemic states, with startling overlappings. The early simple conception of Virchow, that under 50,000 white cells is a leukocytosis and over

50,000 white cells is a leukemia, has long since been replaced by a more complicated arrangement. The recent studies made by Love[10] cover the situation so thoroughly that no attempt will be made to enlarge on them. Too often in all these cases the significance of ulcerations of the tonsils, acute stomatitis, and necrotic areas of the cheek is overlooked, and we may soon see a gangrenous stomatitis, a noma, or agranulocytosis in what we at first considered a simple tonsillitis, or possibly Vincent's angina, for the Vincent's organism is a constant inhabitant of the damaged tissues, and by blocking the vessels adds to the necrosis.

In order to get the background and to see the pathological pictures in their true light, it might be well to review briefly the modern view of the production, destruction, and reproduction of the various components of the blood. In passing, it is well to note before we take this up, the group of deficiency diseases, such as scurvy, sprue, purpura, and hemophilia, which may present a similar clinical picture, but have an entirely different background and are amenable to an entirely different type of treatment, though in this connection, it is possible to consider all these blood dyscrasias, whether due to deficiencies or toxins, as simple non-specific bone marrow reactions. Embryologically speaking, the first cells of every series are almost identical, so that embryologists are divided into three classes: monophylists, or unitarians; the dualists, who divide cells into two types (this group includes Ehrlich); and the trialists, who find three divisions (and in this group is Schilling). Briefly reviewing the red cells, there must be borne in mind the method by which a normal red cell level is maintained. Quoting Osgood and Ashworth[11] in their recent *Atlas of Hematology:* "The red cells are produced by the marrow at a rate which constantly balances their constant disintegration in the blood stream and their removal by the reticulo-endothelium tissue, especially the spleen." However, the liver and the reticulo-endothelium structures elsewhere in the body take part in this process. The duration of life of the red cells is apparently about eighty days, so that each day $\frac{1}{80}$ must be reproduced to offset their destruction, and if there is an increase in the rate of loss, such as by hemorrhage, malaria, or hemolysis, there must be an increase in the rate of production, or a decrease in the rate of destruction. A loss of blood from external wounds tends toward depletion of the store of blood-forming elements, and we find this, naturally, in the loss of blood from surgical procedures. The production of anemias, or pathological

changes in the red cells, may come about from lack of production and supply to the marrow of the anti-pernicious anemia principle. Pernicious anemia itself, so far as we are concerned, may be classed as a deficiency disease, and its symptomatology, except hemorrhage, is not often presented in our field.

Anemias due to infection are the normocytic type, probably caused by either depression of marrow activity or interference with the maturation of the normoblasts, and this is especially prone to be found in the hemolytic type of invasion. It apparently shows very few changes in the field of otolaryngology that are not easily recognizable, but in the aplastic anemias, results may be manifested in the fields of stomatology or otolaryngology before being recognized by either the internist or the general surgeon. This, as well as the agranulocytoses, may be produced by any of the benzol ring, by dinitrophenol, by X-ray, or by radium, and probably also by the sulfanilamide group. It presents one curious manifestation, showing the interrelationship of bone changes to the blood picture; the bone marrow may be entirely replaced by bone. This, of course, is of little importance to us, but the converse, the cystogenic type of bone disease, is sometimes found by the otolaryngologist, and one example will be shown. The symptoms of aplastic anemia presented are very similar to those of the leukemias, agranulocytosis, and purpura hemorrhagica. Diagnosis can really only be made in some instances by sternal puncture, which will show immature cells in the leukemias and a few or none in the aplastic anemias.

The earliest recognized and the most thoroughly studied of these groups is that of the leukemias. The work of Love was mentioned before, and the picture of enlarged glands, pallor, greatly swollen tonsils, and hemorrhage, if we mistakenly remove them, is familiar to all. The pseudo-leukemias of infancy do not concern us so much, and the leukemic infiltrations in the inner ear are not included in the scope of this paper. It is not our purpose to go too far in this division, but it is interesting to bring out one fact, and that is, that extreme cases may be practically considered as malignant tumors of some cell in the leukocytic series. The association of fever and the symptoms of infection have caused many to believe that the acute leukemias are infectious in origin and different from the chronic, but gradations may be found. The history of the leukocyte throws some light on the lesions found in the mouth and gastro-intestinal tract in leukemia. White cells are produced in the red bone marrow and are destroyed and thrown out into the diges-

tive tract, chiefly through the mucous membranes of the mouth and gums. As Pepper[12] points out, "This, perhaps, suggests that the constant involvement of the gums, mouth and pharynx in leukemia is analogous to the splenic enlargement of the anemias with increased blood destruction. Of course, local differences in anatomy and physiology influence very much the distribution of such manifestations. Such local determinants are not the same in the ear as in the mouth, nor the same in the eye as in the tonsil. The mouth and the colon as frontiers constantly exposed to bacterial invasions have more in common than the mouth has with its neighbors, the eye and the ear. One is, therefore, not at a loss to understand why the same necrotic lesions often occur in mouth and in colon in agranulocytic leucopenia and seldom, if ever, in eye or ear. The old German view of the analogy of the mouth and colon, and the resemblance between the tonsils and Peyers' patches, comes to mind.

Leukocytosis is, of course, nothing but an increase in the total leukocytic count beyond the normal for the age group, and leukopenia is nothing but a decrease in the leukocyte count below the normal limits or a change confined to one type of cell. Leukocytosis is usually the term employed, when, in fact, the chief increase is in the neutrophiles (polys). In our field, leukocytosis is found in nearly all inflammatory conditions with normal reactions and a not overwhelming invasion. In our intracranial complications the usual rise is over 20,000, with 85% to 95% neutrophiles. In infectious mononucleosis, the count may run 10,000 to 40,000 with a lymphocyte count of 75% or above, but a poly count of 20% or less. Our tendency is to take the primary blood count for granted and after an initial check-up, to lose sight of later changes, or even to overlook them. Our original leukocytosis may be rapidly modified by significant changes and prove merely to be a phase or a transition state.

Leukopenia, usually with an increase of staff cells, is found in many sinus and throat infections, and is a danger signal, for the normal thing to expect is a leukocytosis. Certain systemic diseases, for instance, typhoid, through their toxins, inhibit activity of the bone marrow, and leukopenia is an almost certain accompaniment. The aleukemic leukemias are embraced in this field, and a fatal case of hemorrhage from a peritonsillar abscess, developing during the aleukemic stage, is afterward to be reported. Mild degrees of leukopenia are frequently found in many upper respiratory infec-

tions. Granulopenia results when the virulence of the infecting organism is far greater than the resistance of the individual, or rather, the productivity of the bone marrow. Kracke[12] holds the view that granulopenia is probably a result of lowered resistance in places easily subject to secondary invasion, such as the mucous membrane of the throat.

Infectious mononucleosis apparently is a distinct disease separated from the mononucleosis syndrome, which probably is a phase in the agranulocytic picture. Early observers considered that agranulocytosis and mononucleosis were identical, but recent works show that mononucleosis is a separate entity. The transitional conditions shade down from one into the other so gradually that, clinically, separation is difficult. However, blood changes in infectious mononucleosis occur, so that sera from these cases agglutinate sheep cells in a higher titer than do sera from normal individuals, as brought out by Paul and Brunnel[13] in 1932. One explanation of sheep cell agglutinins in the sera of these cases is that the agent of the disease serves as a heterogenic antigen. This separates infectious mononucleosis from the mononucleosis frequently present in one stage of the others. A series of cases clinically similar, but without the presence of these agglutinins, has been considered glandular fever, and separated in this way from infectious mononucleosis. Clinically, the throat manifestations are very similar to the other cases in this group, but with more cervical adenitis. As noted before, laboratory findings in mononucleosis show an average to a high leukocyte count of which about 75% to 90% are lymphocytes, although there may be a transient leukopenia, and fenestrated nuclei in the lymphocytes are often found. As stated above, no specificity can be demonstrated; local determinants on the basis of a blood dyscrasia are responsible for the different or similar picture, as the case may be.

Reverting to the cinical manifestations, these are strikingly similar in their onset, which brings up the question again; does that infection cause the blood disorder, and then does the disordered and disorganized blood-producing system, with its intricate checks and balances, break under the strain, resulting in mass molecular death, sloughing of soft tissue and bone, and often rapid somatic dissolution? It is true that these lesions are not confined to the upper respiratory and upper digestive tracts, and that they may be found around the vulva and elsewhere, but the majority are problems for our field, and finding the answer may lead us

past the laboratory pathologist into the field of embryology. As it stands, no definite division can be made, and any attempt, as yet, at scientific accuracy, would be premature, but to quote again from Pepper, "Hematology does not find its field of greatest practical value in the identification of rare blood dyscrasias, but rather in the study of disorders encountered daily by the physician, the surgeon, and the specialist."

The cases presented will be taken from an allied group, showing the different clinical aspects that we find, but hematologically belonging chiefly to the blood dyscrasias in the mononucleosis category, although not strictly covered by the current term, infectious mononucleosis, and in fact including leukemia, but all presenting various mouth, throat or sinus lesions.

Many years ago, the baffling and hopeless aspects of these cases intrigued the writer, as it must have intrigued others, and the following case, with its fatal outcome, still is vivid.

CASES

Case No. 1 — *Aleukemic leukemia:* A Porto Rican bandsman was admitted to the Naval Hospital at Washington, just at the close of the World War. His chief complaint was sore throat, but a thorough blood study, under the direction of Admiral Stitt, (MC) U.S.N., then a Captain in command of the school and active in laboratory work, developed the diagnosis of aleukemic leukemia. This sore throat went into abscess formation on one side. An assistant, called during the night, because the man was choking, incised the abscess as an emergency measure (though instructions were not to open it surgically). An immediate improvement occurred, followed by a closing up of the abscess. Emboldened by the successful outcome of the first experience, the assistant incised the abscess wall again, and continuous slow hemorrhage ensued, which could not be checked, and after a few days the man died.

Case No. 2 — *Agranulocytosis:* The first personal case of agranulocytosis recognized came about eleven or twelve years ago; the fiancée of a young Naval Hospital Corps man, who developed what appeared to be the usual tonsillitis, progressing on the right side to a boggy swelling resembling quinsy. On incision, no pus was found; merely grumous-looking material, and no improve-

ment occurred. In fact, the mild cervical adenitis became more marked. The general condition was, up to this time indicative of a follicular tonsillitis; but a more complete study showed only 2,000,000 reds and 800 whites, the latter dropping to 300. At about that period, Vincent's angina was a bogey, so, because these organisms were present, treatment was along that line. In a few days, the right tonsil sloughed out as cleanly as a dissection. Massive lung abscess, which ruptured spontaneously, evacuating immense quantities of pus, followed, and then lung gangrene, and death. Agranulocytosis was just getting recognized and the belated diagnosis was made. A preceding influenza had caused the use of some of the coal tar products, but no definite or convincing history was obtained either then or later.

Case No. 3 — Mrs. H. H. M., *Mononucleosis Syndrome* (Drug Causative?): Following a cold, the last part of March, 1937, an acute pansinusitis developed, which was followed by an acute otitis media. Physical examination negative otherwise. The family history and previous history were of no import except for a mastoid operation in 1929. There was no history of taking drugs, X-ray examination showed opacity over both antra, anterior and posterior ethmoids, and to a lesser extent the frontals. Remaining sinuses appeared clear. Examinations of the mastoids showed many of the cells on the right side to be absent (operated side). The left showed slight haziness of the cells nearer the attic, but no evidence of cell wall disintegration. Bilateral puncture of the antra was done and a double paracentesis; no other surgery.

Blood Count:	(1)	(2)		(3)
Leukocytes	11,800	12,800		12,000
Polys	82	68		59
Lymphs	18	32		36
			Eosins	3
			Mast	1

Culture from Ears:
Staphylococcus citreus anhemolyticus
Gram positive bacilli present (not classified)

Culture from Nose and Throat:
Streptococcus anhemolyticus

She was hospitalized, and under supervision of an internist and chief of laboratory, was given prontosil at first, then prontolin.

The symptoms subsided locally and she was discharged from the hospital. After a few days she returned to the office complaining of feeling very badly. Blood examination by Dr. Cajigas showed an absolute neutropenia and changes in the red cells. This was believed to be a drug reaction due to prontosil. She was immediately rehospitalized, given intravenous glucose and small blood transfusions, as well as pentnucleotide. At this stage she developed a gingivitis with superficial ulcerations. After several months, her blood picture had not returned to normal.

Comment: While it is possible that this case would have developed the neutropenia without the sulfanilamide, the sudden change following its administration and the changes in the erythrocytes makes it probable that this should be classed as a drug reaction. This illustrates the rapidity of blood changes and the necessity for frequent studies.

Case No. 4 — Mrs. E., *Mononucleosis Syndrome; Antrum and Jaw Involvement: Negative family history*, negative previous personal history, with the exception of a recent upper respiratory invasion of unclassified type, influenza-like in symptoms, which led to a broncho-pneumonia involving the left lung chiefly and which left as an aftermath considerable depression and debility, which was followed shortly by an abscess of the ischiorectal type which was incised and drained and which necessitated frequent subsequent cauterizations. For two weeks preceding her visiting the office, she complained of stuffiness of the left side of the nose, which she considered a cold, and violent headache, which lasted about two weeks, and were treated by the usual coal tar products. At the end of about two weeks, considerable loss of weight, fever, swelling around the left side of the face appeared. On rhinological examination, free pus was found on the left side of the nose, and blocking of the left antrum. The blood count showed an absence of polys, whites 6,000, L.L. 10, small 88, endos 2. Immediate hospitalization was executed, no operative procedures undertaken. After a few days, swelling and tenderness occurred around the left upper molars. Dr. Lynch, a dental surgeon, with mere finger manipulation, removed three molars which left a cavity leading into the antrum, which was found to be filled with thick, bloody material. No repair work was undertaken, small blood transfusions were found the most effective. Later, a dark hemorrhagic spot appeared in the soft palate above the tonsil fossa, but involving the upper

part of the anterior pillar. It looked like an immediate slough, but it subsided and a slow recovery was made, but it took more than three years before the polys had returned in any appreciable percentage. Even now, with the slightest infection, they will drop to around thirty per cent. Pentnucleotide was found less effective than blood transfusions and liver extract. Without careful blood tests, this case would have been mistaken for the usual acute antrum and active surgical intervention would no doubt have been followed up by mass sloughing, no repair, and possibly a fatal outcome. The effects of the coal tar products are problematical.

Case No. 5 — Master C., *Leukopenia, Gangrenous Gingivitis, Osteomyelitis:* Family history negative, previous history negative. Following playing in a pool or puddle, he developed what was considered a cold, followed by pain and swelling around the left side of the face, which was considered merely an acute antritis, though there was bogginess and swelling over the gum and upper left antral region, and X-ray evidencing the usual cloudiness and opacity on that side. An incision of the Luc-Caldwell type was made in my absence and drainage was established. Free pus was found beneath the periosteum, but after opening the bone, little was found in the way of destruction. No recovery had occurred in the next few days, and on my return, marked sloughing occurred in the premaxillary segment, developing into an abscess in the floor of the nose and septum. A tremendous swelling and edema around the left eye, with purplish discoloration and apparently beginning gangrene followed. A small incision on the left orbit, resembling a beginning Kronlein, was made and a drainage tube inserted beneath the lower lid, where free pus was found and sequestrated bone removed. Smear and cultures in the mouth showed masses of Vincent's organisms and pathological sections, made by Dr. Cajigas, showed soft tissue necrotic, blood vessels occluded and the clinical picture was gangrenous osteomyelitis. Later, blood stream invasion occurred with metastases to the bones of the left ankle and foot. Then small osteomyelitic areas on the frontal developed, and recent sequestra extruded. The blood picture in this case is very similar to the others mentioned, a low blood count, and now has an extremely low poly count.

Treatment: Small blood transfusions were given with the best result. Staph vaccine was used because of the metastases of bone, with but little result. Bacterial cultures always showed mixed

growth. After a year's illness, the patient is able to return to school, though small sequestra occasionally appear.

Case No. 6 — Mrs. B.: (Noma) *Gangrenous Stomatitis:* Case reported in detail some years ago in the Archives. Woman about 35, excellent previous health, no history of drug taking. Condition first noticed as an ulcerated area above the left molars, which had been preceded by gingivitis, which was considered a Vincent's angina, and a sodium perborate pack applied. At the time first seen, there was marked swelling on left side of the face. Radiograph showed cloudiness of left antrum; upon exploratory puncture, antrum was found filled with grumous-looking fluid. Blood picture is what we now would recognize as an acute bone marrow deficiency, marked leukopenia, white cells around only 2,000, though polys were about 75%. This case ran a very spectacular course, finally ending up with a gangrenous area over the left cheek, extending to the left antrum, and finally spreading back to involve the ear and scalp. This ulceration was painless until nearly the last. Locally, at that time, while practically every application recommended was tried, the best results were obtained by the use of sodium citrate compresses. Maggots were used at one time to remove the sloughing tissue, but because of disastrous psychic effect on the patient, their use had to be discontinued. Blood transfusions, on the other hand, gave excellent psychic relief and created a feeling of well being. Irradiation of the long bones and other measures in vogue at the time were without result, and the patient finally succumbed.

Comment: At the time, we were grasping vaguely at the etiology of this case. Reviews of the literature at that time afforded us but little assistance. In the light of our present knowledge of bone marrow deficiency, many unexplicable details are now clear, though it must be admitted that our therapy has not advanced much more than we attempted then, in our stumbling efforts, to find relief.

Case No. 7 — Mr. X., *Paget's Disease:* A man above 50 years of age has had this condition for several years and is now living, a year after. There is no known method of treatment and it is simply presented to illustrate one of the rare bone conditions that may be confused in the field of otolaryngology. The X-ray examination of the skull, giving the characteristic "nigger wool" appearance, in

an advanced case would make the diagnosis clear, though if seen early the protruding eye, nasal discharge, and opaque antrum on transillumination and X-ray could be confusing.

While there may be disputes regarding the entity of these various conditions, the common origin of many of the symptoms seen in the group of blood dyscrasias cannot be gainsaid. Anemia, leukopenia and thrombopenia are present as primary or secondary changes, whether the arrest of maturation of cells in the red bone marrow is caused by drug ingestion or bacterial toxin. Even the aberrant cells of the leukemias may not be seen at first in the aleukemic stages. Naturally, the treatment would be divided into two parts; the local, which is probably covered very well by the method of Marriott,[14] in which the mouth is sprayed with a mild anesthetic and then cleansed with hydrogen-peroxide, about 50%, and then again thoroughly swabbed with a 10% citric acid solution. Besides this, everything has been used from caustics, oxidizing agents, bacteriophage, and, in the case we mentioned, necrotic tissue was removed at one time by maggots. All local treatments may be summed up briefly as cleanliness, opening small pockets and abscesses, removing necrotic tissue, and no surgery beyond that. The general treatment, aiding and controlling the marrow weakness by taking off some of its work, is paramount. Blood transfusions, preferably small and repeated, the use of leukocytic extract, pentnucleotide, and adenine sulphate, the removal of deleterious drugs and toxins, and the direct stimulation of bone marrow by irradiation of the long bones are all beneficial. The use of leukemic blood in transfusions has been reported by H. E. Bock,[15] of Berlin, with favorable results. Theoretically, it sounds plausible, for the equivalent in white cells of many ordinary transfusions can be gotten in one. The use of turpentine injections to produce artificial abscesses has been advocated with a view to stimulating leukocytosis. This is reminiscent of our medical ancestors with their use of setons, and the practice of veterinarians today.

Conclusion: Quoting Bethell,[16] "The response of the bone marrow to infection is dependent upon its pre-existing functional state, as well as upon the nature and severity of the infectious process."

On this basis, the bacterial invasion, the drug, the deficiency or possibly the environment of the individual, such as a prolonged sojourn in the tropics, produces a bone marrow response. This is manifested in the blood stream, if the action is on the leukopoietic elements by a change in the nature and number of the white cells, the preponderance of immature forms, or if in the elements that produce the red cells, by a corresponding change in the erythrocytes. While no specifics have been evolved as yet, our best treatment has been blood transfusions, and this has the merit of being logical, for in this way stress and strain have been kept away from the already weakened and overworked bone marrow. The local reaction, due to the local determinants, is very frequently first manifested clinically in the field of otolaryngology and stomatology and is illustrated in its several phases by the clinical pictures we have presented.

BIBLIOGRAPHY

1. Fenton, R. A.: *The Physiologic Approach to Otolaryngology.* J.A.M.A., 106:1775-1776, May 23, 1936.
2. Furstenberg, A. C.: *A Clinical and Pathological Study of Tumors of the Nose, Pharynx and Mouth of Teratological Origin.* Trans. Am. Laryngological Assn., 55:128-138, 1933. Also, Trans. Am. Laryngological Assn., 58:231-248, 1936.
3. Mosher, H. P.: J.A.M.A., 107:942-947, Sept. 19, 1936.
4. Madison and Squier: *Further Observations on Relation of Amidopyrine to Acute Primary Granulocytopenia.* Trans. Am. Therapeutic Society, 34:76-82, 1934.
5. Benjamin, J. E., and Biederman, J. B.: *Agranulocytic Leukopenia Induced by a Drug Related to Amidopyrine.* J.A.M.A., 107:493-949, Aug. 15, 1936.
6. Witts, L. J.: *Prophylaxis and Treatment of Agranulocytosis.* British Med. Jour., 1:1061-1063, May 23, 1936.
7. Boughton, T. H.: Dept. of Internal Medicine, U. of Illinois.
8. Kracke and Parker: *Relationship of Drug Therapy to Agranulocytosis.* J.A.M.A., 105:960-966, Sept. 21, 1936.
9. Dameshek, Wm., and Colmes, A.: *The Effect of Drugs in the Production of Agranulocytosis with Particular Reference to Amidopyrine.* Journal of Clinical Investigation, 15:85-97, 1936.
10. Love, A. A.: *Manifestations of Leukemia Encountered in Otolaryngologic and Stomatologic Practice.* Archives of Otolaryngology, 23: 173-221, Feb. 1936.
11. Osgood and Ashworth: *Atlas of Hematology.* San Francisco, Stacy, 1937.
12. Pepper, O. H.: *Hematology and the Blood Dyscrasias in Relation to Diseases of the Eye, Ear, Nose and Throat.* Ann. Otology, Rhinology and Laryngology, 42:87-94, 1933.
13. Van Ravenswaay: New England Medical Journal, 211:1001-1004, Nov. 29, 1934.

14. Marriott from (6).
15. Bock, H. E.: J.A.M.A., vol. 109, Sept. 11, 1937. (Report from Frankfort Med. Society).
16. Bethell, F. H.: *Bone Marrow Dyscrasias.* Journal of Lab. and Clin. Med., 120:362-374, 1934-35.

DISCUSSION

DR. GEORGE M. COATES, Philadelphia: The essayist has just given us a broad general survey of interesting hematological conditions with nose and throat manifestations and included several case reports.

For purposes of discussion, I shall merely comment on some of the larger aspects of the paper, drawing such conclusions as observation, experience and reasoning may lead me to.

From the very beginning of my study of otolaryngology, I was impressed with the theory, or hypothesis, that many conditions present in the nose and throat were local manifestations of a general condition (D. Braden Kyle), and that while local measures might be of value, it was most important that the general body balance should be reestablished. I was cognizant of the fact that septic infections in the nose and throat often did produce a motley group of pathologic changes in the great vital organs, in the blood, in the joints, ears, intracranial structures, eyes, etc. Time and again the blood-forming organs are likewise severely damaged by sepsis and infectious processes from the upper respiratory tract (or other foci of infection) as was well pointed out by the essayist. In this, as in any other infection, the amount of ultimate structural and physiologic damage to the blood-forming organs will depend upon the virulence of the infection and the resistance of the host. There may be a number of other clinical conditions that at present are not fully understood, but in which future studies will ultimately reveal that a focus of infection in the upper respiratory tract has been the first cause, or that it has accentuated another cause.

As regards the blood dyscrasias, it seems to me that these conditions often show some symptoms and signs in the nose, throat and sinuses, which are merely part of the clinical picture of the dyscrasia. To say, therefore, that sepsis in the upper respiratory tract is the cause of these dyscrasias would be to reason in a circle.

It is the discussion of the subject of mononucleosis with its manifestations in the nose, throat and sinuses with which we shall now concern ourselves in particular. This entity has been thoroughly reviewed by Dr. Trible, and certain points of interest and comments will be stressed again. This disease, also termed "glandular fever," is rarely fatal, but contagious and causes an enlargement of the lymph glands. In children, it may occur as the "acute" form, with enlarged cervical glands, sore throat, and pyrexia as the main findings. As stated, it is contagious, often epidemic. As a rule, no rash is noted. In the adult, however, it may manifest itself in one of three forms, though one type may dovetail into the other. The three types described are

the glandular, the anginose and the febrile. The glandular type is characterized by malaise, fever and headache, which is the period of invasion, and lasts about three days, followed by an eruption lasting about 10 to 14 days, the type being a maculopapular rash. As this fades, the glands enlarge. When the glands swell the temperature rises again, subsequently remitting. The spleen is often enlarged. The anginose type is the one wherein sore throat with membrane formation is the outstanding finding. This type is said to resemble diphtheria, but there are a number of methods of differentiating the two. The febrile type resembles typhoid fever.

We see, therefore, what difficulties the otolaryngologist is confronted with when he sees a reddened, congested pharynx, with a patient toxic, and running a variable febrile course. A blood count will at once give us very important information. In infectious mononucleosis or grandular fever, we shall find a total white blood count of (12,000 to 30,000) or *less* (leukemia is 500,000 to 1,000,000) with *lymphocytes and monocytes* forming 6 per cent or more of the number. Occasionally, lymphocytes are in the majority, and at other times monocytes prevail. It is very important *not to rely upon one blood count*, for in the beginning a neutrophil leukocytosis may be found, while later the neutrophils will gradually disappear and a true lymphocytosis will manifest itself. Therefore, it is wise to make several blood count determinations. It is also important to get a correct reading of the cells, for often transitional or immature cells appear, and the result will not be absolutely true. There is still much confusion at the present time as regards nomenclature. Many hospitals report the hemoglobin in percentage. This, of course, varies as to the type of determination made. Thus, percentage in the Sahli differs from the Tallquist, and the Dare, Haldane, Oliver, and the Williamson-Newcomer all report different percentages when the hemoglobin is reported, let us say, as a content of 17 grams per 100 cc. of blood. A uniform method of classifying the blood count and the character of the cells will go far toward giving the clinician and otolaryngologist the true reading. The same technician should repeat the count.

Inasmuch as the treatment of mononucleosis is purely symptomatic, and as no specific remedy has thus far been found, the gentlest measures applied to the upper respiratory tract in order to render the patient more comfortable are all that is necessary. Strict isolation of the case is imperative because of the contagious nature of the condition. Bed rest for the febrile period is most important. The strength of the patient should be kept up by means of adequate and optimum nutrition, high caloric diet, sufficient sleep and rest, and tonics, because the convalescence is protracted, as a rule, and weakness, lassitude and debility may persist for months.

The throat symptoms (or those of the nose and sinuses) will disappear as soon as the general infectious mononucleosis has disappeared. We should appreciate that many conditions in the body will cause symptoms in the nose and throat, and when we are confronted with *any* throat symptoms, we should at once ask for a blood count, and it should be repeated as often as necessary, for the count may reveal whether a neutrophil or lymphocytic leukocytosis exists, or perhaps a leukemia or agranulocytosis. Let me once more stress the blood picture from 12,000 to 30,000 — rarely beyond that; a 60 per cent or more of lymphocytes or monocytes; little or no anemia, and a normal platelet count.

Dr. John J. Shea, Memphis: Dr. G. B. Trible is to be congratulated upon calling attention to the hematopoietic responses possible as the result of infections in the field of our specialty. The relationship of the anginas and the hematopoietic system is uncertain, for in one instance an infection of the throat may be the initial lesion and in the other a terminal manifestation of a hematopoietic disease.

As Dr. Trible remarked, in some of the late cases of the leukemias, you will have lesions in the mouth that are the result of the hematopoietic response and not the response of the result of the lesion.

The older physiologists considered the blood as a special form of connective tissue having a fluid matrix. The modern conception is that the blood is a mixed secretion, the formed elements of which are the result of parent cells living outside the blood stream. There exists a barrier which prevents immature blood cells from entering the circulation. The physiologist cannot explain its action or what happens when it fails, thus allowing immature cells the privilege of the blood stream. The toxins of the various anginas may stimulate or paralyze the hematopoietic system, similar to roentgen rays, small doses stimulating, massive doses paralyzing.

This is important in the distribution in the blood stream of your polymorphonuclears. There is a normal distribution, and when this is disturbed it is an indication of a false response of your hematopoietic system.

The bone marrow houses an emergency supply of polymorphonuclear leukocytes, and can deliver them into the circulation when needed. If the surplus is consumed and the newly formed cells are not sufficient for the demand, immature forms will be found in the circulation. That is, you will have a greater number of your segmented cells in your differential count as compared with the mature cells. I want to call your attention to the monocytes, as the monocyte count in your blood picture is always important, for a true monocyte increase is a good sign, as it is a stage of response.

The most common causes for the presence of immature cells in the blood are: (a) hemorrhage, (b) diseases that destroy blood cells, (c) various substances that act as stimulants to the hematopoietic system and (d) hyperplasia of the hematopoietic tissues.

The infectious of the throat are characterized by the influences they exert on the hematopoietic system, and belong to the third class. On the other hand, they are primary diseases of the blood that produce lesions in the mouth, and it is difficult to separate the borderline cases.

The laryngologist has an opportunity to study the relationship of the anginas and the hematopoietic response in the following diseases: Agranulocytic angina, acute infectious mononucleosis, Vincent's angina with a lymphoid leukocytosis, and acute lymphadenosis.

The anginas are capable of producing four types of changes in the blood: (1) polymorphonuclear leukocytosis, as seen in follicular tonsillitis and septic throats; (2) mononuclear leukocytosis, as occurs in infectious mononucleosis; (3) leukopenia with a decrease of granulocytes — agronulocytic angina — and (4) leukocytosis with a relative hypogranulocytosis, as seen in the patients suffering from a generalized Vincent's infection.

Dr. Trible is correct in his contention that the patients who present agranulocytic responses to drugs are rare, or else we would encounter

countless cases. The administration of sulphanilamide must be watched for both action upon the hematopoietic system and a combination with hemaglobin in the presence of a sulphate. The latter produces a sulphemoglobinemia after the second administration of 10 cc. of prontosil solution. Investigation revealed that she had been taking large doses of sedatives.

Case one of Dr. Trible's series was similar to a case seen in consultation following a tonsillectomy. The man was forty-eight years of age and was reported as having had very large glandular type of tonsils. The wound continued to ooze after his operation and when his blood was studied, the surgeon was shocked to find that he had operated upon an acute lymphatic leukemia.

Each of the doctor's patients should serve as a reminder to us that every patient should be studied thoroughly and that a routine complete blood count is necessary.

This is the type of blood picture that is a monocyte increase, but it is a lymphatic monocyte increase. It is not a leukemia. It is a case reported in there of a patient who had an acute Vincent's, in which the dentist had extracted too many teeth. There was a response of 22,000, with 90 per cent lymphocytes, and went on to a fatal termination.

This is the type of picture that we see in a true mononuclear leukocytosis, and it is a little exaggerated because we do not see as many of the large monocytes as the artist has pictured here.

Of course, this is the type of field you will see after making two or three searches in a true case of agranulocytosis, the agranulocytes, and here and there just an occasional agranular cell.

(Several slides were shown.)

DR. G. B. TRIBLE, closing: I want to take the congressional privilege of extending my remarks in the record, and I simply want to thank Dr. Coates and Dr. Shea for their very generous and very illuminating discussion.

TREATMENT OF CHRONIC LARYNGITIS SIMPLEX

LOUIS H. CLERF, M.D.

PHILADELPHIA

THE TREATMENT of simple chronic laryngitis, a common nonspecific chronic inflammatory disease of the larynx, presents one of the most difficult problems in the entire field of laryngology. The large number of predisposing and exciting etiological factors, a majority of which are found outside the larynx, justify Kyle's dictum that the laryngeal changes often are a "local manifestation of a systemic condition."

The belief that chronic laryngitis can be cured by local measures applied to the larynx alone obviously is based on a lack of knowledge of the underlying pathological changes and a failure to appreciate the importance of etiological factors in the causation of disease. Before considering treatment, it would seem important, therefore, to consider briefly the pathological alterations in the laryngeal structures and to discuss causal factors.

The changes in the tissues vary depending upon the character of the lesion, the intensity of the inflammatory reaction and its duration. They may vary in different individuals due to certain constitutional reactions. Although this cannot be proven conclusively, there undoubtedly is a curious tendency in some individuals towards excessive fibrous tissue proliferation. When the irritation is continued, the hyperemia and cellular infiltration of the mucosa and submucosal layers are followed by organization of the inflammatory exudate with an increase in connective tissue elements. Although this commonly involves the entire larynx it usually is most marked in those structures exhibiting the greatest activity, namely, the vocal cords, ventricular bands and interarytenoid area. With increased irritation there is thickening of the epithelium with generalized fibrotic hypertrophy of the soft tissues and musculature and changes in the circulatory and glandular structures. This may ultimately lead to greatly thickened and rounded vocal cords, hypertrophied ventricular bands and fibrosis of the muscles with interference in normal approximation, vibration and tension of the

cords. Treatment, to be effective, therefore, must be directed towards relieving the tissue changes which interfere with normal laryngeal function as well as removal or correction of the cause.

Etiology. — With the exception of abuse or excessive use of the voice, the causes of chronic laryngitis are found outside the larynx. Any condition capable of producing prolonged diffuse hyperemia of the larynx may be a cause of chronic laryngitis. These may be roughly classified as due to infection or irritation and to effects from systemic conditions. Under infection may be considered diseases of the upper air and food passages and of the tracheobronchial tree. The irritative causes would include smoke, dust and gases as well as excessive use or abuse of the larynx. Among systemic conditions would be included the effects of alcohol, defective elimination, gout, hepatic or cardiorenal disease and hypertension. More than a single factor is commonly operative in a majority of cases. For example, with acute laryngitis, if he goes on vocal rest and takes care of himself, as an acute case of laryngitis should, he will have a spontaneous recovery. If, on the other hand, he insists upon using his larynx continuously and he continues to smoke, recovery will not occur as promptly as it should, and he will pass into the acute, into the subacute, and then into a chronic case of laryngitis. It is also important to recall that in syphilis, tuberculosis and carcinoma of the larynx there is present a chronic laryngitis which, in the earlier stages, must be reckoned with diagnostically.

Treatment. — In treating a chronic nonspecific inflammatory disease one must treat the patient as well as the disease. Alleviation of symptoms is important. Although the effects of local treatment are temporary, they will aid in enlisting the patient's interest and assistance. Ultimate success in treatment depends upon the detection and removal or correction of the etiological factors. The salesman who talks constantly and improperly, smokes incessantly, drinks freely of alcoholic beverages, eats irregularly and travels at night in a Pullman sleeper is interested less in the advice given concerning correction of his habits than in the local measures to be employed in securing relief from his laryngitis.

Before proceeding with treatment, it is imperative that a diagnosis of chronic laryngitis is made. Many cases of carcinoma of the larynx are erroneously labelled as chronic laryngitis until the

disease becomes advanced. A diagnosis of chronic laryngitis should be made *only* after the exclusion, by appropriate studies, of all conditions simulating chronic laryngitis.

The *local measures* employed in treatment depend upon the severity of the symptoms and the laryngoscopic findings. Every effort should be made to decrease the chronic congestion of the larynx. Three dont's, namely, *don't talk; don't smoke; don't use alcohol,* should be enforced. Vocal rest, preferably absolute, is an important aid; in the excessive voice user it may be necessary to rest the voice for several months. That brings up the important problem of the individual who must make his living by using his larynx, and there may be a happy medium that can be established in a general way, but in a general way vocal rest is important. Smoking is irritating to the larynx and tracheobronchial tree and should be interdicted, particularly if indulgence has been excessive. Smokers of cigarettes usually assert that inhaling aids to get rid of thick secretions, particularly in the morning. In the case of a man who comes in to see me because of hoarseness and a sensation of a foreign something in his throat, he smokes three to four packages of cigarettes in twenty-four hours, and when he stops smoking the cigarettes in two days his sensation of a foreign something in his throat disappears and he feels a lot better.

Smoking does irritate the larynx, although when you tell smokers of cigarettes to cut out smoking, they usually insist that inhaling cigarette smoke aids to get rid of thick secretions, and that is one of the troubles with symptoms in chronic laryngitis. It aids to get rid of thick secretions, particularly in the morning, and they want you to make exceptions so they can smoke in the morning. Alcohol, which normally produces hyperemia of the larynx, should be discontinued. These measures may suffice in a simple case of laryngitis if the cause can be removed. In the more obstinate case, additional measures will be necessary.

Local medication directed to the larynx may be administered by inhalation, application and instillation. Gargles do not reach the larynx. Sprays and inhalers, in the hands of patients uninstructed in their employment, usually are not effective. Direct applications to the vocal cords are prone to traumatize the mucosa. Instillation by the indirect method constitutes a very effective means of laryngeal medication. Medicaments are employed to secure a cleansing, astringent, sedative or antiseptic effect. Thick, sticky secretion may be gotten rid of by employing an alkaline solution or preferably

normal salt solution either as a spray or by indirect instillation. Inhalation of compound tincture of benzoin or of menthol with the aid of a vaporizor is useful in relieving irritation. Instillation of an oily solution as Mono-P-Chlorphenol, grains one to the ounce of liquid petrolatum, or menthol, grains five to fifteen to the ounce of liquid petrolatum, by mirror laryngoscopy giving about one cc., is useful. Oil of pine has been recommended highly. The topical application of silver nitrate, zinc chloride and iron perchloride has been advocated. I have not found it desirable to employ these.

I fully appreciate that these few suggestions regarding local treatment do not take into account the known fact that every laryngologist has at least one pet formula that he uses in his cases of laryngitis.

Obviously these measures are employed to give temporary symptomatic relief. Their use will produce little change in the altered laryngeal tissues. The causes must be sought for and removed. This often is obscure and requires a comprehensive study of the patient and an inquiry into his habits, occupation and environment.

Abuse of the voice is a common cause of chronic laryngitis. It often is observed in those shouting or cheering, in prolonged excessive use, as in speaking or singing; or in improper use, as in those training to become singers. Frequent attacks of acute laryngitis or failure to rest the larynx until the inflammation has subsided are common causes.

In a search for causative factors the nose and pharynx should be examined for obstructions. The upper air passages, the ears, and the mouth, teeth and gums should be searched for foci of infection. The faucial and lingual tonsils often are the seat of disease in chronic laryngitis and may serve as a reservoir of infection. Remarkable improvement in obstinate chronic laryngitis has been observed following removal of diseased faucial tonsils in the absence of other demonstrable causes.

I had that impressed upon me very forcibly in a patient whom I treated for some time. He was an obstinate patient. When he left me he went to someone else and the other doctor took out his tonsils, and he came back to exhibit himself. His laryngitis cleared up as if my magic. I always look at their tonsils now.

Disorders of the nasal passages, accessory nasal sinuses and pharyngeal tissues are the most important and potent factors in

the etiology of chronic laryngitis. This probably results from direct extension of the tissue changes, direct infection of the larynx with infected or irritating material and interference with normal nasal function.

If there are symptoms of dysphagia the esophagus should be investigated. Patients with organic stenosis of the esophagus, cardiospasm or pharyngeal diverticulum often have overflow of secretions and food particles into the larynx, particularly while sleeping. This may be responsible for a chronic laryngitis and tracheitis.

Chronic laryngitis may be secondary to chronic tracheobronchitis or bronchiectasis. Every form of chronic pulmonary infection including tuberculosis must be excluded.

Coincident with these studies one should secure a careful history with reference to the patient's habits, notably as regards smoking and drinking. The occupation should be inquired into with reference to exposure to dust, fumes, gases and cold, hot or dry air. Atmospheric conditions in themselves are not important factors unless these are marked. Frequent trips from a heated to a cold atmosphere without adequate protective clothing may ultimately become an important causal factor.

In the more obstinate cases, particularly during advanced life, cardiac, renal or hepatic disease, gout, autointoxication and blood dyscrasias should be considered as possible factors.

In a comprehensive study, it is evident that one or more probable exciting or predisposing causes will be discovered. The close correlation of these renders it difficult, even hazardous, to express a positive opinion regarding the outcome of treatment. Correction of a nasal obstruction or removal of diseased tonsils may not influence chronic laryngitis in a public speaker. Changing one's occupation from a dusty to a dust-free environment may not be followed by improvement. One must evaluate one's findings in a given case and be guided by experience and the patient's insight to his problem.

A change in climatic environment may be helpful. Dry air, windy localities and cold winters should be avoided. A warm, balmy climate is preferable. Sea air may be beneficial. Continental writers recommend a course of treatment at a spa. It is assumed that the general health would be carefully guarded.

In adductor paresis, the result of fibrosis of the intrinsic laryngeal muscles, massage of the neck, administration of strychnine or iodides, and the use of faradic current have been recommended.

Caution must be exercised in the use of iodides which usually are contraindicated in laryngeal inflammation.

In marked hypertrophy with redundancy of the mucosa of the vocal cords removal of small polypoid masses or stripping of the cord as practiced by Loré may be resorted to. These procedures should be followed by prolonged periods of rest.

To summarize briefly, chronic laryngitis is a nonspecific inflammatory disease, the causes of which are many and varied. Treatment consists of employment of local measures to the larynx and removal of the cause. Although the effects of local treatment are temporary, their use is important. Etiological factors must be sought for in the patient's habits, occupation, environment and general health. Improper use of the voice and disorders of the accessory nasal sinuses, nose and oropharynx are the most important factors in etiology. The results in treatment are dependent upon finding the cause and upon its correction or removal.

DISCUSSION

DR. JOHN M. LORÉ, New York: We all agree that in cases of chronic laryngitis, there are a number of cases that do not respond to the usual nonsurgical method. What about these cases? We reviewed a series of such cases some time ago and tried out certain surgical procedures. I should like to call your attention to the observations made.

For instance, the cases of laryngofissure where you have the reticular band, the vocal cords and intervening cord, watch the healing process, how that deficiency fills up and as time goes on how the epithelium grows in from the sides, until after weeks of healing you find that you have what we are pleased to call a regeneration of mucosa.

You may have had the experience of removing a benign growth, such as a polypus, and being a little vigorous, you may have removed some of the cord with it, inspecting the result with dismay and feel perhaps you had done a tremendous amount of damage to the larynx. What a grand and glorious feeling it was several weeks later to note in the repair there had been a regeneration of mucosa with no disturbance of function.

Predicated upon this observation, a series of experiments were carried out on cats to note what did happen when mucosa from the vocal cords was removed.

The technique for these experiments was the utilization of cats. Various amounts of mucous membrane in the vocal cords were removed. The cats were observed over a period of time, three, four, five, six weeks and two

months, and then the cats were destroyed for other purposes. New vocal cords were observed.

The only difference that we were able to note between the normal cord of a cat and that which regenerated after removal was the matter of thickness, and I dare say that had we waited sufficiently long this would have been overcome, too.

What should constitute the indications for surgical procedure? First of all we must assume that a thorough and intelligent rational method of nonsurgical treatment has been carried out, with no results or little results. In the second place, we must have some pathological changes in the mucosa, polypoids, papillomas, or whatnot. That is absolutely necessary in order to create an indication for removal of a cord. In addition, we have that process called the hyperkeratotic papillomas. In this connection, while I am on the subject, I should like to mention that some years ago at the New York Academy of Medicine, I went on record that I considered it a pretentious healing. Subsequently, I went a step farther and said that hyperkeratotic papillomas are carcinomas. I have had six cases in which eventually we were able to demonstrate malignant changes in the tissue, if not early, always in later specimens.

(Slides were shown.)

Dr. M. F. Arbuckle, St. Louis: For the reasons pointed out by Dr. Clerf, and with whose expressions I agree, this disorder might very properly be called chronic laryngitis "complex," rather than "simplex." The more we study the local and the general manifestations of the disorder, the greater, it seems to me, are the difficulties in carrying out successful treatment. One of the first and most frequent difficulties is that of establishing with certainty the exact diagnosis. Not infrequently, or one might say, in general, the patient with chronic laryngitis simplex is an individual who is busily concerned with making a living and, of necessity, indulging in those very activities which so often should be curtailed. It is difficult to persuade the busy man to stop long enough to have a biopsy specimen taken, so that any malignant disease may be ruled out. Much less is he inclined to stop or curtail his activities when no such danger is at least apparent. This arrest of activities includes the correction of systemic disorders which (as has been pointed out) do play such an important part as etiological factors. It also entails attempts at correction of injurious habits and the institution of proper hygienic measures in many instances. Thus, it is apparent that the situation is "complex" indeed, since the disorder is one with such variety and combinations of causes and in which the difficulties of successful treatment are increased because the diagnosis is not always easy, treatment interferes with activities, and the situation is not sufficiently grave to warrant or justify interrupting daily routine.

In the case in which there is a tendency to "excessive fibrous tissue proliferation," we usually find the patient is a busy man who uses alcohol and tobacco freely, and his voice to excess; and since this is his daily life he will not give it up. It has been my experience that in such individuals the gag reflex and the degree of irritability of the pharynx is great, sometimes de-

feating our best efforts at mirror examination. Because differential diagnosis is so important, direct laryngoscopy should be carried out when indirect laryngoscopy is unsatisfactory. If this procedure were faithfully carried out and proper biopsy study made in questionable cases, many a tragedy would be averted. Apropos the question of the danger of stimulating growth, or actually causing neoplastic change by taking biopsy specimens, I would like to go on record as one of those who feels that biopsy is so important and so necessary at the same time (when properly performed), so free of harm, that when indicated it should be carried out without the slightest hesitation and by direct laryngoscopy.

In general, as treatment goes, I think the milder the remedy applied locally the better. Probably the most important local treatment, along with the removal of chemical irritants, such as tobacco, alcohol, dust and other fumes, is voice rest, and this surely is the one most difficult to have carried out. Systemic disorders, such as pulmonary suppuration of any type with cough, kidney disease, occasionally hypothyroidism, suppuration of the upper respiratory tract, etc., must have proper attention. The local application of strong or harsh remedies, such as silver nitrate or zinc, etc., is mentioned only to be condemned.

Dr. Louis H. Clerf, closing: I simply wish to thank the contributors for their remarks on the presentation, and also to agree with Dr. Arbuckle as to the complexity of simple chronic laryngitis.

AUDIOMETERS AND HEARING AIDS*

AUSTIN A. HAYDEN, M.D.

CHICAGO

AUDIOMETERS. For the practicing otologist, the audiometer[1] is an office electrical instrument for measuring hearing. Direct or alternating current may be used. It measures hearing loss in decibels (db) at calibrated frequencies (pitch) and supplants the old methods in which the voice, watch, acumeter, tuning forks, rods, bells, whistles, etc., were used. The examiner may now increase, decrease or hold stationary the intensity of any frequency on the audiometer for any length of time desired. The decay of the tuning fork always limited its usefulness and was outstanding among errors inherent in the old methods.

The full hearing range that we have taught and have been taught, from 20 to 20,000 cycles (vibrations) per second, and for which former testing equipment was manufactured, is not covered in its entirety by any audiometer exhibited at this meeting. This is largely because of excessive manufacturing costs and doubtful clinical utility. However, the older "instruments of precision" can and should be used when necessity arises or comparisons are desired.

In general, audiometer findings are plotted so that frequency runs *horizontally* across the chart from 64 to 8192 or over and is calibrated in octaves, half-octaves and/or octave letters. Sweeps cover all or part of the octave calibrations and go far beyond these pitches to as high as 16,000 cycles per second. Intensity is measured in decibels (db) between the thresholds of audibility and pain and is charted *vertically*. The ordinary telephone receiver transforms the electrical energy of the audiometer into sound cycles for air conduction through the external auditory canal. For bone conduction through the mastoid, a specially constructed receiver is provided.

* This work was facilitated by an expense grant from Sonotone Corp.

1. "A Clinical Audiometer is an instrument for measuring the acuity and range of hearing." Tentative minimum requirements for acceptable Audiometers, Council of Physical Therapy, A.M.A.

Additional equipment now available includes maskers, microphone amplifiers for speech articulation tests, plans and specifications for sound proof rooms (an excellent example of which Dr. Horace Newhart is exhibiting at this meeting), rubber receiver cups for use where such rooms are impractical, and specially prepared charts.

Hearing Aids. An electrical hearing aid raises sound level for transmission by air through the external auditory canal to the drum and by bone — thanks to Lieber for the bone conduction receiver — through the mastoid to the inner ear. They are portable, semi-portable and stationary. The first uses small single or multiple dry cells; the second, either large dry cells or line current, or both; and the last, line current only. This paper deals only with the portable aids because they are the most widely used.

Portables are amplified or non-amplified. They are specially adapted telephone hook-ups, consisting essentially of a receiver (ear piece), transmitter (microphone) and battery. In the amplified set, an amplifier or booster with its second circuit is added. Cells may number from one to four and the battery delivers 1½ v. to 6 v. Too few or worn out cells prevent sufficient amplification.

The frequency range of portables lies principally from 256 to 2,000 cycles per second where most conversational tones come. Some amplification is possible as high as 3,500 or 4,000 cycles per second. By altering the construction of the transmitter the sound can be "peaked" at one or more frequencies; by changing the characteristics of the receiver, another "peak" is obtained. Thus sound may be "peaked" or intensified quite effectively within three divisions (low, middle and high) to fit approximately the hearing loss shown on an audiogram. Amplification can also be spread out quite evenly over a fairly broad area. Thus, the electrical and mechanical characteristics of the transmitter, receiver and booster when used in various combinations afford literally hundreds of different hearing aids. How many are actually practical is a very wide open question.

Response Curves of Hearing Aids — Audiograms of Hearing Aids — Master Hearing Aids. By passing the audiometer's calibrated tones into the hearing aid's transmitter and measuring the output with a decibel meter, the overall "tone characteristics" of the various arrangements of component parts can be plotted

into "response curves" of hearing aids. The actual change the hearing aid makes in the patient's hearing can be ascertained by attaching the audiometer's ear piece to the transmitter of the patient's hearing aid and charting the audiometer reading of the patient in the usual way. This is the *audiogram of the hearing aid.*

The audiometer may be compared to the retinoscope. The latter determines both degree and position of errors of refraction. The former locates both position (frequency) and degree (intensity) of hearing loss. "Tone islands" correspond roughly to astigmatic errors of refraction and indicate the position (frequency-pitch)[2] at which the intensity (loudness) of the hearing aid should be "peaked." Just as the trial frame enables the patient to look through the various lens combinations indicated by the retinoscope, likewise, the deafened patient can actually *hear through* the half-dozen combinations indicated by the otologist's audiogram. Carefully worked out[3] speech articulation tests determine the combination best suited to the individual requirements.

This great improvement in fitting has been effected by the development of what, for want of a better generic name, may be called the "master hearing aid." All but one of the four largest manufacturers fit from the findings of this instrument. This firm uses audiograms only, which are made in its own sales rooms. Its "audiometric analysis" is advertised to be for hearing aid fitting only and not for the diagnosis of ear disease. Two consider audiograms unnecessary and depend entirely on these instruments to make their fittings. The fourth uses its own master hearing aid in addition to an *otologist's* audiogram, which it believes to be *essential.*

Just how the vacuum tube works in the audiometer, how sound is amplified, how the attenuator sweeps the entire intensity range between the thresholds of hearing and pain, how to define bel and decibel, and how to comprehend sound in terms of drum surface pressure requires highly technical knowledge. The hearing aid presents similar problems. Otologists may well leave such matters to

2. *Pitch* is that subjective quality of a sound which determines its position in a musical scale.

   Loudness is that subjective quality of a sound which determines the magnitude of an auditory sensation produced by that sound.

   American Standards Association, Oct. 15, 1936 — Bulletin Acoustic Terminology.

3. Need for speech articulation tests well treated in British Medical Research Council's report of Committee on the Physiology of Hearing — Special Series Report No. 219, London — His Majesty's Stationery Office.

the Bureau of Standards, the American Standards Association, American Medical Association's Council on Physical Therapy, and to their professional associates and designated consultants.

TESTS ON AUDIOMETERS. Inasmuch as unnecessary "minimum requirements" increase the cost and thereby, to some extent, limit the use of any apparatus designed for wide clinical use, it is desirable to determine the value of such requirements. In audiometers, pure wave form, which does add greatly to construction costs, has been considered necessary, especially by laboratory workers. To determine its real value in actual practice, thirty moderately hard of hearing people were tested with four audiometers.[4] Three of these — X, Z, and 2A — probably meet Council requirements, while Y has an admittedly imperfect wave form. Western Electric 2A was arbitrarily taken as a standard for comparison because of its long-established (but unfortunately far from sufficient) use and the literature it has created.

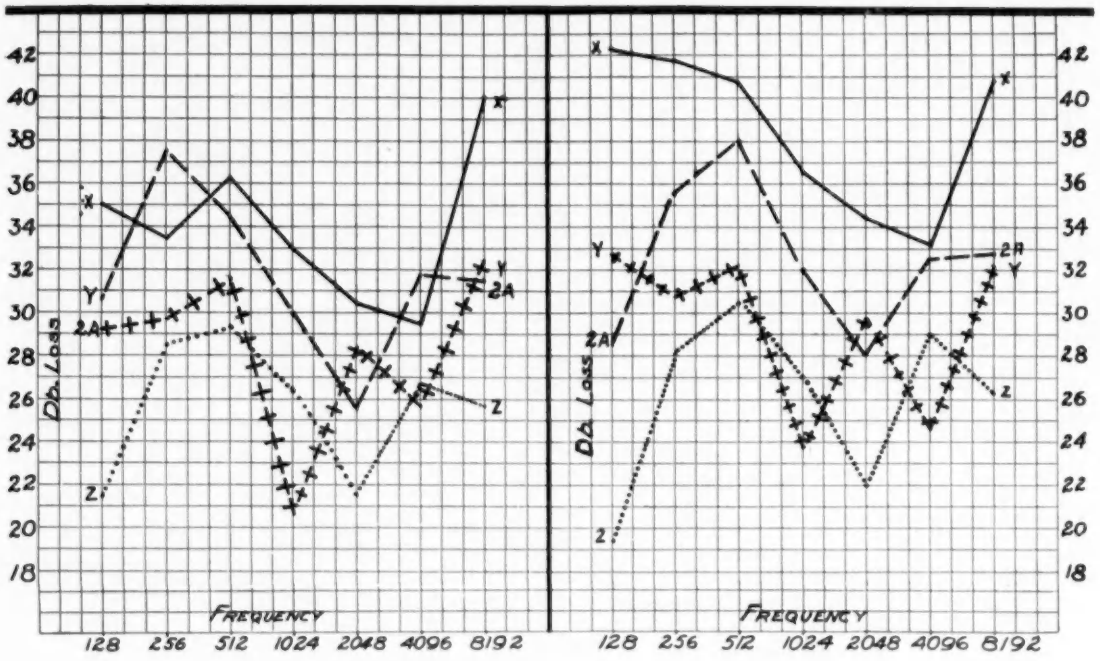
The four audiometers were placed in a small inside room of an otologist's office. The doors and windows in this and adjoining outside rooms were closed during the tests. The average noise level was then reduced from 50 to 20 db. A thick rubber hood around the air receiver with a similar cup over the opposite ear further lowered the noise level, but to exactly what extent was not determined.

Audiometer X is provided with dials for octave (128 to 8192) as well as sweep frequencies (2500 to 17,000). Intensity is calibrated in steps of five decibels. It is provided with an automatic self-compensating pointer. Audiometer Y has octave, octave letter and sweep frequency range from 65 to 8192 with attenuation from 0 to 100 db. at all frequencies. Audiometer Z has two dials, one for sweep frequency marked with eight indicated points from 128 to 9847. For bone conduction tests, a ring is placed over the intensity dial for re-calibration.

When being tested, the patient responded by the usual finger or electric signal. The average of a minimum of three readings by air conduction at each of the octave frequencies was taken to be the actual hearing loss and charted as such on the individual audiogram, and recorded. These three readings almost never varied from each other by more than plus or minus 5 db. The tests were

4. X (Sonotone), Y (Marvel), Z (Western Electric 6A), and Western Electric 2A designate audiometers used.

CURVES SHOWING AVERAGE (16 INDIVIDUALS) LOSS OF HEARING
AS MEASURED BY FOUR DIFFERENT AUDIOMETERS (RESULTS OF 896 OBSERVATIONS)



X, Y, Z, and 2A designate Audiometers tested.

Intensity (loudness) is plotted vertically in db;

Frequency (pitch) at octive intervals horizonally across the chart.

Tests at octave letters, as well as sweeps, through the conversation range (256-2048) would quite likely have revealed the existence of many "tone islands."

The figure 896 is obtained by multiplying 4 (audiometers), by 7 (frequencies tested), by 2 (ears), by 16 (patients). Inasmuch as each observation is an average of a minimum of three readings, the individual observations actually total more than 2588.

made by three experienced observers. Rest periods were used as needed to minimize fatigue-confusion of both patient and observer.

Fourteen of the thirty were found to have hearing losses greater than the Western Electric 2A audiometer could measure. For that reason their audiograms were discarded. The hearing losses of the remaining sixteen, as shown by each of the four audiometers, were averaged and plotted. Differences between readings at most frequencies are apparent on the chart. The average difference over all frequencies is readily compiled and is not great.

SUMMARY.

(1) Four audiometers of different makes were investigated to determine their comparative performance for air conduction tests[5] and the need for pure wave form in clinical practice.

(2) Audiometer X consistently showed the highest hearing loss; Y showed a medium, but more erratic, loss; and Z the smallest hearing loss. As compared with the 2A the average differences were plus 5 db. for X; minus 3 db. for Y, and minus 6 db. for Z.

(3) Audiometers X, Y, and Z were capable of measuring greater hearing losses than 2A.

(4) Plus or minus 5 db. were found to be the combined human error factor of observer and patient.

(5) An average "quiet" — not sound proof room — was used for the tests.

CONCLUSIONS:

(1) Audiometers, despite the foregoing variations, furnish the best means of testing hearing acuity.

(2) Any one of the four tested will be more useful in clinical practice than any other means now available for testing and recording hearing acuity.

(3) The lack of purity of sound wave apparently did not introduce any serious errors that were not largely explainable by other causes.

(4) A quiet room is essential. The need for a sound proof room increases as the hearing loss to be tested decreases.

(5) An audiometer should be a part of the office equipment of every otologist.

5. Comparisons for bone conduction will be subsequently reported.

Clinical material obtained from private office practice, the Chicago League for the Hard of Hearing and the out-patient department of St. Joseph Hospital.

* * *

Through its Otologists' Committee on Audiometers and Hearing Aids,[6] the Council on Physical Therapy of the American Medical Association places its facilities at the disposal of Otologists for the purpose of correlating clinical experience, laboratory procedure and manufacturing detail. Council acceptance should become an incentive to manufacturers to construct instruments in accordance with agreed upon clinical requirements and assure Otologists that advertised claims meet required specifications.

DISCUSSION

DR. HORACE NEWHART, Minneapolis: I regret very much that until a few moments ago, I knew nothing of Dr. Hayden's paper. Owing to his illness I had not been able to see it in advance, and so I must come to you entirely empty-handed as far as any organized discussion is concerned. But I welcome the opportunity to say a few words to you concerning a subject in which we are all interested and have been more interested the last few years because of a recognized need on the part of otologists for better methods of detecting slight degrees of hearing loss. In this respect the older methods have proved themselves inadequate because of their inaccuracy, and because of the fact that they consume so much time if carried out according to the best ideas. Even so, their results have been poor compared with what is now obtainable by audiometers even though they are not as yet standardized. I feel that Dr. Hayden's paper has been a most eloquent justification of the fine efforts which are now being made, largely through the instrumentation of the Council on Physical Therapy, to make it possible for the man doing otology to have in his possession a reliable instrument at a moderate cost.

I think that Mr. Carter will tell you that some day these instruments will be possible of standardization because of release of a minimum specification with instructions.

6. This Committee is composed of Doctors George M. Coates, Lee Wallace Dean, Edmund Prince Fowler, Austin A. Hayden (Chairman), Isaac H. Jones, Douglas MacFarlan, Burt R. Shurly, Horace Newhart, and William P. Wherry. Mr. Howard A. Carter is Secretary. Several of the individual members have availed themselves of the services of such outstanding Physicists as Bunch, Fletcher, Hartig, Knudson, and Rogers.

I want to take just a few moments of your time to make a few points regarding this matter of testing the hearing with accuracy.

By merely glancing at this slide, I think you can see why it is that the ordinary use of the whispered voice and even the conversational voice, upon which we have relied so much, are absolutely inadequate under ordinary conditions of use in a quiet place to detect those slight degrees of hearing loss which in the young cause speech defects, retardation in class in school, and the more severe or persistent cause inferiority complexes, and in some cases even peculiar or unsocial conduct has been observed.

These facts have come to us only recently through the wide application, in millions of cases, of the so-called phonograph audiometer that is used in our public schools. As a result we find that there are about 3,000,000 school children who have hearing deficiency, and at least 300,000 of them are unable to hear a whispered voice, and it has proven that we must point out that the phonograph audiometer should never be used as a diagnostic machine purely for whispering purposes.

The next slide will give you an idea as to why it is necessary to detect in these youngsters a slight degree of hearing loss, by this table showing the relative phonetic powers of fundamental sounds as produced by the average speaker. For instance, the energy required or with which the sound of "th" as in "thin" strikes our auditory apparatus is represented by the unit "1", whereas when we go into the vowels, the loudest vowel and the sound most readily perceived but often missed by people with lowered hearing capacity, 68 instances.

We learned recently that speech defects occur most frequently in most cases which have losses for the high tones — that is, they lose out when it comes to catching the sound of the vowel. So the interpretation of speech after all is largely guesswork for all of us unless we are very close to the speaker.

One of the great obstacles to better work in the early diagnosis of incipient ear trouble, especially the loss of high tones, as we find it rather frequently in children with focal infections, is the fact that we are constantly laboring in the midst of a sea or ocean of noise. All of us who work in the Loop District, let us say, or who work in schools, for instance, are constantly interfered with by distracting and masking noises. So we wish to emphasize the great importance of taking measures to overcome the noises that are most interfering. You see what they are even in residential districts where we have forty decibels. Therefore, the test is invalid, and we cannot detect the loss under the level of the prevailing noise.

To overcome this the physician must resort to some sort of acoustic treatment. The attainment of a sound proof room is beyond the present income of the average otologist, but by use of a little acoustics properly employed one can so reduce the noise level that it is workable. It is no longer a menace to good work. This we accomplish in this particular case in a small room by the simple use of acoustic treatment, and the application of a heavy rug, a suspension of the ceiling, and we reduce the sound level by at least 15 or 20 decibels, which makes this sort of work more nearly satisfactory and certainly more nearly accurate.

A Committee on Deafness was carrying on some experiments in the University of Minnesota in an effort to design an economical and efficient

means of overcoming this problem of noise, and in the exhibit here today, to which Dr. Hayden referred, we have a knock-down portable, crudely constructed example or a working model of such a booth. I hope that most of you who are at all interested will take occasion to test it out by seeing how far you can hear a given noise in the presence of the noise that prevails in the room. Then go to that booth and learn how much farther you can hear. You will find that a person with a hearing loss as shown outside the booth of five or six inches is reduced by stepping into the booth and closing the door to such a degree that he hears distinctly five, six or seven feet, and I think one who makes this experiment will be convinced of the necessity, especially if his office is in the downtown district, of taking this means of protecting himself against erroneous work.

As a matter of fact, this is the outside of this booth, and it has this advantage. There is a necessary point in its construction to isolate it from wide radiant floors and walls by the installation of sponge rubber.

This simply shows a preliminary drawing, but it is the thought of this committee to be able to furnish men, after several months when our work is completed, plans and specifications so that members can equip themselves in this way with something that is efficient and yet will be inexpensive. If we do not, we will find that the downtown vendor will be somewhat better equipped than ourselves and we will be discredited by the fact, and I think those fellows know a lot more about the subject of otology than some otologists.

MR. HOWARD A. CARTER (by invitation), Chicago: Doctor Hayden's study is of particular interest to me as Secretary of the Council on Physical Therapy of the American Medical Association. As you know, it is the assigned duty of the Council to investigate and report on apparatus that is recommended to the medical profession for therapeutic and diagnositc purposes. By the cooperation of specialists and consultants, the Council is undertaking the consideration of audiometers and hearing aids.

It seems reasonable to state that in view of the evidence presented by Dr. Hayden, the audiometers that are now on the market cannot be regarded as the final achievement. To improve the situation, it would seem that some authoritative body, such as the National Bureau of Standards, might well establish a standard; for example, an artificial ear for determining the intensity and frequency of audiometers. Duplicate artificial ears might be distributed to various interested bodies. It is interesting to note that instruments X and Z meet the requirements of the American Standards Association, whereas only instrument X meets the requirements of both the American Standards Association and the unadopted tentative requirements of the Council on Physical Therapy for acceptable audiometers. It is highly doubtful that Audiometer Y would meet the requirements of either the American Standards Association or those of the Council on Physical Therapy. Yet, it would seem, if these curves are reliable, that, in general, all three audiometers examined would give information of equal reliability.

DR. AUSTIN A. HAYDEN, closing: First of all, I want to commend and to agree thoroughly with what Dr. Newhart said about testing in sound proof rooms. I want to say that not everyone has his own; nor for other reasons can he secure a sound proof room. For that reason the tests were done in a room that had a noise level as stated.

I am very sorry about the things that Dr. Newhart said about the testing with the phonograph audiometer, for this reason: I feel that there is a great need for diagnostic pure tone quality audiometers, such as have been talked about here today. When Newhart checks the thousands of children in Minnesota that he has with the phonograph audiometer, what are you going to do about it? There isn't any proper equipment scattered throughout the length and breadth of the country for follow-up work, and we are just playing with these phonograph audiometers to a very large extent until that equipment is present in otologists' offices. That is the plea that I make today and the plea that I hope you will heed.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

PRESIDENT LEE W. DEAN, St. Louis: The purpose of this panel discussion is to summarize the known facts relative to sigmoid sinus infections. In order to bring this information to you, we have gathered a galaxy of stars, each an authority upon the particular subject assigned him. We will discuss the matter frankly. It will include the pathology, routes of infection, anatomy, variations in the sinuses — all of which may be a part of the clinical picture.

In the event you disagree with a speaker, or his conclusions, you may use the card which Dr. Myers has given you and on this card state your impression, and give it to Dr. Myers, who will hand it to me for proper distribution.

We, ourselves, not only desire to have a clear conception of the accepted procedure of diagnosis and treatment of this condition, but we want each one of you to have the same opinion when the panel has been completed.

It is easily conceivable that some one of us here may not understand a point that has been made and we will be privileged to raise that question with the speaker across the table. In the event that there is anything not clear in your minds, I wish that you would make use of the card, hand it to Dr. Myers, and it will be answered by one or more of the speakers.

For instance, to show how the panel is going to work, I am going to ask Dr. Lillie a question.

I have heard you say, Dr. Lillie, that sometimes when you suspected an infection of the sigmoid sinus you operated on the mastoid first, and that, after you had completed your operation, you perhaps might deem it advisable to ligate the jugular, and if you did consider it advisable, you went ahead and ligated the jugular.

Now, Dr. Lillie, will you tell me how, after you have been operating in an infected mastoid, you can enter the neck in a clean field and ligate the internal jugular without infecting the neck?

Dr. H. I. Lillie, Rochester, Minn.: That would seem simple enough. It can be done in two ways. You can rescrub and redress, and if you anticipate the necessity for going into the neck, you will have a second set of instruments. Another way in which it could be done in an institution is to have a second team who would do it. I have no hesitancy about approaching the mastoid first, and whether I tie the jugular vein afterwards depends upon what I find in the mastoid and sigmoid sinus.

One must remember that the neck field, if the infection has extended into the jugular or into its surrounding glands, is also infected. It is practically impossible to have a surgically clean neck wound whether the vein is attacked first or not for those reasons.

Does that answer your question?

President Dean: Yes, indeed.

I am going to ask all the gentlemen about the table to ask questions when they differ with a speaker. If the members about the table do not differ with a speaker when a statement is made, the audience has the right to assume that we all agree with that statement. Consequently, we are under obligation to differ and to defend our premise, if not in accord.

So, with this explanation, we will proceed with the panel.

The first speaker will be Dr. George L. Tobey, Jr., of Boston, Mass., who need not necessarily confine himself to his subject, unless he wishes.

Dr. Tobey: May I have the privilege of reading my manuscript, Dr. Dean, subject to interruptions and questions?

President Dean: You may do anything you wish, Dr. Tobey.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

* * *

Diagnosis

GEORGE L. TOBEY, JR., M.D.

BOSTON

THE DIAGNOSIS of thrombophlebitis of otitic origin is dependent upon a critical consideration of the whole symptom complex as present in the individual case. An existent or preëxistent infection of the middle ear is presupposed. The otitis may have been so slight as to have escaped observation or, if observed, may not have demanded surgical interference.

Before speaking of septic thrombophlebitis, I wish to draw your attention to acute septicemia of otitic origin, occurring without a thrombosis. It is well known that during the course of an acute otitis media and mastoiditis, there may be a direct invasion of the veins draining the middle ear and mastoid, with extension into the general circulation without the formation of an intermediary thrombus. Indirect extension may also occur by way of the emissary veins. The picture is that of a sudden pre- or postoperative onset of acute symptoms of septicemia; there is a sudden high elevation of temperature followed by an equally rapid decline, occurring at intervals, often accompanied by chills; there is an increased leucocytosis and possibly a bacteriemia may be present. Having ruled out other sources of infection by a careful and painstaking differential diagnosis, of which I shall speak later, ligation of the jugular vein has often resulted in an immediate cessation of the symptoms and with no subsequent metastasis.

Many of you have had this experience and have been disappointed that you found no macroscopic evidence of thrombosis.

PRESIDENT DEAN: Dr. Tobey, may I interrupt? Do I understand you to say, sir, or perhaps not to say it directly, but given a child with tonsillitis, an acute purulent nasal sinusitis, and an infection of the middle ear, do I understand that you think that such a patient presents a picture of sepsis, positive blood culture, with metastatic involvement of the joints, perhaps in the fingers and elsewhere, without having the septicemia secondary to thrombophlebitis of the sinus? Could this come from infected tonsils or infected sinus, or must it come from the ear?

DR. TOBEY: No, Dr. Dean; I said, "subject to a very careful differential diagnosis." I will speak later about the differential diagnosis.

PRESIDENT DEAN: But then as I understand it, you do feel that the picture of septicemia may result from an acute upper respiratory infection.

DR. TOBEY: Without doubt, decidedly.

DR. SAMUEL J. KOPETZKY, New York: May I interject here? Where there is no tonsillitis, and where you have drawn the picture of a septicemia with a positive blood culture from the ear alone, is it not possible, Dr. Tobey, that you are dealing with minute thrombi in the subepithelium tissue? I say this because later I will show you specimens which will demonstrate this viewpoint; and, therefore, I take a slight exception to the statement that there is no thrombus in the tissues of the middle ear when you have your sepsis. You may have thrombosis, although macroscopically you can not see such a minute lesion.

DR. TOBEY: I am speaking now of a demonstrable thrombus in the vessel.

DR. KOPETZKY: Well, demonstrable both macroscopically and microscopically; do you mean macroscopically?

PRESIDENT DEAN: May I interrupt, gentlemen, for just a minute? Dr. Hartmann, will you tell us something of the difficulty of differentiating between a pulmonary lesion that is responsible for sepsis, and sepsis secondary to tonsil infection in patients who have

infected mastoids, and may I ask you if you have seen cases where the infection comes from the tonsils, which have been diagnosed as septic thrombophlebitis of the lateral sinus, but have recovered without any particular attention being paid to the ear? Will you please discuss that question from the pediatrician's standpoint?

Dr. Alexis F. Hartmann, St. Louis: You ask one of the hardest questions first. I am sure we have seen septicemia, associated with ear and mastoid infection, simulating clinically sinus thrombophlebitis, get well without any particular surgical intervention; and I am sure, also, we have had a number of children, who, during the course of their otitis media and mastoiditis, became so ill with the remittant type of temperature, with fluctuations of as much as 8 to 10 degrees Fahrenheit, that we suspected strongly involvement of the lateral sinus, but came to the conclusion eventually that their trouble was due to a pneumonic process rather than to a lateral sinus phlebitis.

Now, in helping us to incriminate, so to speak, the lung, as Dr. Dean has asked, and to exonerate the sinus, there are a number of things which we must consider. As far as blood culture goes, it is my strong feeling that positive blood cultures may be seen early in the course of any acute upper respiratory tract infection, or lower respiratory infection, but unless a large vessel like the lateral sinuses is involved, the cultures tend to become negative in a short time; and, furthermore, one does not often find a streptococcic septicemia in the course of what looks like a typical pneumonic process unless that process happens to be embolic in origin and secondary to a phlebitic process. So it is such findings in the blood cultures, repeated day by day, which I think help us more than anything else, Dr. Dean, in evaluating the lateral sinus involvement as the chief cause for sepsis and in exonerating the lung as anything but a secondary factor.

President Dean: Thank you very much, Dr. Hartmann.

I will apologize to Dr. Tobey for anticipating his discussion, but we should emphasize the difficulty of making a diagnosis of involvement of the sinus where there is an upper respiratory infection associated with one in the ear.

Dr. Tobey: A phlebitis involving the lateral sinus and the jugular vein, with or without an obliterating thrombosis, will cause symptoms of a less explosive type. Phlebitis readily extends along

the course of the jugular vein; however, it will not extend below a point of ligation, a fact to be considered by the conservatist. In the general consideration of the symptomatology of septic thrombophlebitis of the lateral sinus on which the diagnosis rests, there are four distinct types of temperature charts:

Type A. The accepted picture of acute sepsis with very sudden elevation of several degrees and equally sudden remissions to normal, or even subnormal. This may occur several times within a few hours, or but once or twice during the twenty-four. I have seen several cases where it has occurred at intervals of at least twenty-six to forty-eight hours.

Type B. The temperature at no time shows an elevation of more than one or two degrees, but a persistent irregularity, with a usual evening rise, although it may vary several times during the twenty-four hours. To my mind, in all probability this type of temperature takes place during the formation of a mural thrombus.

Type C. An elevation of two to three degrees, which does not remit to normal but shows slight remissions and elevations from 100° as a minimal. This may persist for several days and then return to normal. This type nearly always recurs after remaining at normal for a few days. This, I believe, as a rule takes place during the formation of the obliterating sinus, and intermissions of high temperatures, with or without chills, in all probability are due to the breaking down and absorption of the thrombus.

Type D. A normal temperature. It is not an uncommon experience in performing a mastoid operation to find a definite sinus thrombosis, and occasionally a broken down sinus wall and fistula, the patient having at no time while under observation shown a temperature. In a series of seventy-three cases, this occurred eleven times, in three cases the sinus wall having been completely necrosed.

PRESIDENT DEAN: Dr. Tobey, do I understand you to say that sometimes when you operate a mastoid upon an ambulatory patient, without fever and without leucocytosis, you actually find an abscess in the sinus, with a fistula in the wall and pus coming into the mastoid? How do you explain that?

DR. TOBEY: We have a series of seventy-three cases in which this occurred eleven times.

PRESIDENT DEAN: What is your explanation of it?

DR. TOBEY: I have no explanation.

DR. KOPETZKY: I can explain it because I have found it, too. I explain it in the fact that in the process of the formation of the perisinus abscess a sufficient walling off has taken place to protect the system generally. The rupture of the sinus wall and the egress of the pus from the interiorly situated septic portion of the thrombus leaks out into this abscess, which is walled off from the general system, and thus protects the body from systemic reactions. This is my opinion of the mechanics of the lesion, and its asymptomatic course.

DR. LILLIE: May I interject a thought? Is it not this observation that has led some observers to say that for that reason the thrombus in the sinus need never be operated or the jugular ligated?

DR. KOPETZKY: Their observation of the lesion is correct, but the conclusion as to surgical therapy is incorrect.

DR. HARTMANN: I think there is another point, too, that we should bear in mind here. One can have organisms entering the blood stream and still have the patient do quite well for a period of time. For instance, recently we had a boy of about three, who for nineteen successive days had positive blood cultures, for hemolytic streptococci, without the development of any metastatic foci, and a temperature of a rather steady type without much swing; to look at that child and to look at his chart and to examine his blood picture generally did not suggest strongly the presence of septicemia, yet for nineteen successive days the blood cultures were positive.

DR. LILLIE: Was that a hemolytic streptococcus?

DR. HARTMANN: It was a hemolytic streptococcus.

DR. KOPETZKY: In these cases where you have the type just described, it is a primary bacteriemia, and the primary bacteriemia is clinically insignificant as against the period after the primary bacteriemia, the period of no bacteriemia; and then there comes on the second bacteriemia, which is pathologically significant. That

was brought out experimentally, I believe, by Dr. Stengel some years ago in working on experimentally produced thrombosis of the saphenous vein of a dog.

PRESIDENT DEAN: May I suggest that in our series of latent cases of thrombophlebitis of the lateral sinus — if I may use that term "latent" — at Iowa, one-third of the patients had papill-edema. In our series at St. Louis, one-fourth of the patients have had the edema of the optic nerve. Consequently, I am of the opinion that in every case of chronic infection of the mastoid, or infection of the mastoid that has persisted for over ten or twelve days, a fundus examination is always in order.

Pardon me, Dr. Tobey, for interrupting. Will you proceed?

DR. TOBEY: In all four types, the pulse usually follows the temperature and has been found of little diagnostic value. These great variations in the temperature chart indicate clearly that the temperature can not be considered a definite diagnostic symptom. Its presence indicates infection, but its absence or its atypical characteristics do not preclude an infectious thrombosis of the sinus.

The temperature chart must, however, always be taken into consideration in forming the conclusions as to diagnosis.

True rigor or chills occur in less than 50 per cent of the cases. A large majority of patients will complain of a fleeting chilly sensation accompanied by sweating.

Headache, when present, is seldom described as pain. Rather, it is a dull sensation of pressure which the patient is unable to definitely localize. This is accompanied by drowsiness from which he is readily aroused, but which immediately recurs, suggesting a typhoidal picture. This toxemia has a peculiar characteristic in that there are intermittent periods of exhilaration and feeling of well-being followed by periods of depression.

Dr. Dean, I feel that perhaps not as frequently now as in days past, you have had patients referred to you or you have been called in for consultation on cases in the general ward which had been under treatment for typhoid fever, perhaps, and yet you found that they had an infected ear with a sinus thrombosis.

PRESIDENT DEAN: Yes, Dr. Tobey, but living where we do, we have another disease, namely, malaria, which is a not uncommon occurrence. Is that not true, Dr. Hartmann?

DR. HARTMANN: Yes.

DR. KOPETZKY: Isn't there the one distinction between the malaria and the beginning stage of typhoid to be found in the euphoria which is more or less marked in the streptococcic general infections?

DR. TOBEY: Dr. Kopetzky, I am referring, perhaps, to the faulty diagnosis of the general practitioner and not to the otologist, because these patients are the ones whom you find in the general hospital who have been admitted on the supposition and have been treated as typhoid fever.

PRESIDENT DEAN: My first mastoid, in a small town of 8,000, had a brain abscess, and my second one had a septic thrombophlebitis of the lateral sinus, and had been treated in the ward in the hospital for typhoid fever by a person representing himself as a specialist in internal medicine. So these things do happen.

DR. TOBEY: The hematological picture is that of systemic infection. There is, as a rule, moderate increase in the total white blood cells, an average of 15,000 in a large series of cases. A higher cell count is suggestive of meningeal or other complications. The differential study is of importance as an index of the individual's resistance to the prevailing infection. As the patient improves, the immature neutrophiles disappear. A progressive reduction of the red cells and hemoglobin in the presence of sepsis indicates a hemolytic infection.

Bacteriemia, when present, is conclusive evidence of a generalized systemic infection, and when taken into consideration with the other symptoms present is an aid in diagnosis, but it must be borne in mind that it may be present in practically every acute infection, such as pneumonia, acute endocarditis, erysipelas, pyelonephritis, pyelitis, etc. The pyelitis, of course, is more especially found in children. A negative culture does not preclude the presence of infection. A recent survey of sixty-five cases (Meltzer) showed fifty patients with negative blood cultures, and yet thirty of these patients were found to have thrombosis of the lateral sinus. These cultures were taken in series, not depending upon one culture.

DR. KOPETZKY: For practical purposes for the use of men who have not available at their hands good laboratory facilities to take blood cultures — and one must incidentally remember the very many factors which tend to produce a negative culture — hemogram studies taken serially will give them very corroborative evidence of a blood-stream infection; namely, if they do serial blood studies, which are available anywhere, then they will find that the progressive loss of hemoglobin is an indicator that there is something destroying that hemoglobin, and since you have a hemophilic type of infection present in most instances, the deduction is obvious, and this finding saves us from taking of a blood culture where the results of a bad or a poor laboratory diagnosis might put the patient in jeopardy and delay or prevent an operation that actually might be necessary.

DR. TOBEY: Then you will agree with me that you are not going to wait for a positive blood culture before you operate?

DR. KOPETZKY: I never do, provided other evidence permits a diagnosis.

DR. TOBEY: Yet in some of the articles that you read that statement is made fairly definite.

DR. KOPETZKY: I am not responsible for them.

DR. TOBEY: In thrombosis of the venous sinuses, there is usually some degree of meningeal reaction which is demonstrated by an increased cerebrospinal pressure and an increase in cells and globulin in the spinal fluid. At first these cells are polymorphonuclear, but as the reaction subsides, they become predominantly lymphocytes. However, the only absolute determinant of meningitis is a positive culture of the fluid and a biochemical study.

Edema or infiltration, with tenderness on deep pressure over the area drained by the mastoid emissary vein, were the most constant objective symptoms found in a large series of cases, and they were present in practically every case of mural or complete thrombosis of the sigmoid sinus. These indications are quite distinct, and readily differentiated from mastoid edema and tenderness. They never extend forward over the posterior border of the mastoid. In other words, it is a primary osteothrombophlebitis of the emissary vein.

The same findings are present just below and posterior to the mastoid tip due to blocking or infection of the posterior condylar vein which usually enters the sinus just before it forms the jugular bulb. (This vein is not constant.)

Many writers have stressed the importance of tenderness along the course of the jugular vein. However, this is rarely observed, and when present is due to infection of the deep cervical glands and seldom, if ever, is it an indication of jugular vein infection.

I should like to know Dr. Dean's reaction and experience on that statement.

PRESIDENT DEAN: I am going to ask Dr. Lillie to answer that question.

DR. LILLIE: I think that the observations that the doctor has made are essentially correct. However, I just had an experience with a neck that makes me believe that inflammation around the jugular vein in the sheath can cause severe pain. I reported two cases of jugular vein thrombosis in which shooting pain to the ear was a very prominent symptom, in fact the only symptom. The signs were quite indefinite. A recent experience I had in ligating an external carotid artery for a nose bleed was interesting. Each time that I touched the bifurcation of the carotid, the patient complained of a very severe shooting pain to the ear. That explained the symptoms of the pain that I had seen associated with the jugular vein thrombosis. The inflammation within the carotid sheath could very well do that, but I think it is essentially correct to say that the tenderness along the course of the jugular vein is due to the inflammatory involvement of the glands, and that these glands, as we all know, make difficult sometimes our dissection of the neck.

DR. TOBEY: In other words, Dr. Lillie, you would not put too much stress on it in trying to make a decision as to whether or not you had a thrombosis?

DR. LILLIE: No. I think that is essentially correct.

DR. TOBEY: Transient cerebral symptoms, due to stasis, are at times present. Stupor, even coma, have been observed. Aphasia is not rare, and it has been present with thrombosis on the right side. Convulsions, transitory hemiplegia, and cerebellar symptoms have been observed by Weise, Beck and others (Nielson-Courville).

Signs referable to the cranial nerves are numerous. Choked discs have been observed by numerous writers, and in our own observations have been found in 10 per cent of the cases. Nystagmus may be in both directions or it may be rotatory. Sixth nerve paresis is not uncommon. In other words, these transitory meningeal or intracranial symptoms are due to stasis, and, at autopsy, practically no definite pathology of the brain tissue has been found. Paresis of the sixth nerve is due in this type of case, as a rule, to edema secondary to stasis and not to direct infection of the petrous tip or spiralis canal.

Do you agree with this statement, Doctor Kopetzky?

DR. KOPETZKY: Yes, indeed.

DR. TOBEY: Kummel has reported involvement of the ninth, tenth, and eleventh cranial nerves in jugular bulb thrombosis. Varying degrees of ipso-lateral hemicrania are frequent. Jacksonian convulsions have been observed.

Exposure of the sigmoid sinus may reveal a definite perisinuous abscess with no apparent involvement of the sinus wall. Granulations may be present on the sinus, and may extend from beyond the knee to the jugular bulb. In the absence of a fistula through the wall, we have found it impossible to make a definite statement as to any pathologic change which may have taken place within the sinus itself.

The needle puncture is unreliable and not without danger.

DR. KOPETZKY: Dr. Dean, I think that the needle puncture should be warned against, because of the fact, as I shall show in the discussion of pathology, that needle puncture is one factor in an impending case that helps to develop a thrombus. It can not be warned against too much. There are too many men with slight diagnostic efficiency traumatizing the intima of the sinus, and where no thrombosis was present before their injury to the blood vessel's intima starts the development of one.

PRESIDENT DEAN: I should like to ask another question, this time of Dr. Brown, since he will discuss the surgery. In the case of a perisinuous abscess with granulation, as mentioned by Dr. Tobey, what are you going to do with these granulations?

DR. JOHN M. BROWN, Los Angeles: Leave them alone. Leave the sinus alone. I do not open under those circumstances. Wait.

PRESIDENT DEAN: What would you think would be the result if you were to take all of these granulations off nice and clean with the curette?

DR. BROWN: I think that it might be disastrous.

PRESIDENT DEAN: Thank you. Dr. Tobey, will you please continue?

DR. TOBEY: Another point in the needle puncture, Dr. Kopetzky, I have seen the inner wall of the lateral sinus punctured, giving rise to a meningeal infection.

DR. KOPETZKY: I have seen that, too.

DR. TOBEY: The manometric test has been proved to be of undoubted value in demonstrating partial or complete obstruction of the circulation through the lateral sinus and jugular bulb. And, Dr. Dean, if I may be permitted, I should like to go into this very carefully, because I think there is a misconception of the technique of this test.

PRESIDENT DEAN: I agree with you, Dr. Tobey, and I particularly think there is a misconception of the seriousness of the test. I mean to say, many people think it is inexcusable to make a test like this on a child four or five years of age or younger. I think you should go into it very thoroughly, sir.

DR. TOBEY: The test is based upon the fact that increased intracranial pressure is normally propagated throughout the cerebrospinal fluid system and may be measured by a manometer employed in connection with lumbar puncture. Compression of the internal jugular veins causes increased intracranial pressure, with immediate rise in spinal fluid pressure.

PRESIDENT DEAN: May I interrupt just a minute, Dr. Tobey? In order to get the reaction of the pediatrician to this, may I ask Dr. Hartmann what percentage of cases of otitis with suspected involvement of the lateral sinus he makes this manometric test on?

DR. HARTMANN: I can't give you figures, but I can say that very frequently we make spinal punctures and take advantage of

the manometric test at the same time, and if we find everything quite negative we expect such patients to get along perfectly well.

Dr. Tobey: It is now generally considered that absence of a rise in fluid pressure on jugular compression (positive Queckenstedt) indicates spinal subarachnoid block, a most important and early sign in spinal cord tumor. We have seen, in the literature, this test referred to as the Queckenstedt test, and I wish to draw your attention to the fact that while it is based on Queckenstedt's observation, the test itself is entirely different. More recently it has been shown that block may also occur and be detected in a similar manner in the presence of tumor of the cerebellar fossa. In both of these conditions, the evidence on which diagnosis of block rests is absence of rise in pressure below the level of obstruction, the increased intracranial pressure being assumed in single puncture below, or proved by two punctures, one above and one below the level of obstruction. It may be safely said that the principles on which these tests depend and the results of the tests have been trustworthy for the most part. Indeed, the effect of jugular compression on spinal fluid pressure forms one of the routine tests in diagnostic lumbar puncture.

Although the criterion on which a diagnosis of sinus thrombosis is based is the same as that of subarachnoid block, namely, absence of rise in pressure in the spinal fluid manometer, it will be seen that the mechanism by which this is shown is quite different. Instead of an increased head of fluid which can not be transmitted because of obstruction, there is here no elevation of pressure at its source, because of venous obliteration. Also, in sinus thrombosis, we are concerned with a careful comparison of the effects of pressure on each vein separately, not merely with the effect of simultaneous compression of both internal jugular veins, as in the diagnosis of cord tumor.

The technique is as follows: With the patient in the lateral position, lumbar puncture is performed, and the fluid is allowed to run into a glass manometer of 2 mm. bore. The initial pressure reading is noted; also the presence of pulse and respiratory oscillations as evidence of patency of the manometric system. Then, without in any way disturbing the patient, an assistant gently presses on one side of the neck between the larynx and the sternocleidomastoid muscle, compressing the vessels against the transverse processes of the cervical vertebræ.

I think that is one of the reasons that so many of these tests are perhaps incomplete or unsatisfactory. I have watched house officers and I have watched other men make a compression by simply making a compression between the sternocleidomastoid muscle and the larynx without at any time attempting to compress against the transverse cervical processes. It is impossible in the majority of cases to get compression by simply pressing on the vein and the carotid artery. You must go in and bring it back until you compress the vessel against the transverse process of the cervical vertebræ.

During this compression, the operator watches the rapidity of rise of the fluid column in the manometer, the promptness of its beginning, and the full height to which it attains, and, on release of the jugular compression, the rapidity of drop in pressure. The procedure is now repeated on the opposite side of the neck. And then, for comparison, the vessels on both sides are compressed simultaneously. Alternate release will show a rapid drop in pressure on the unaffected side, slight if any change on the affected side.

In a typical case of lateral sinus thrombosis there is a prompt and rapid rise in fluid pressure to twice or three times the initial reading when the internal jugular vein draining the normal sinus is compressed. This pressure rise is maximal, being equivalent to the pressure attained when both jugular veins are compressed. Pressure over the vein draining the thrombosed lateral sinus causes either no rise or more commonly a slow rise of only 10 to 20 mm. in the manometer.

These findings are characteristic of complete obliteration of the sinus. Partial obstruction from mural thrombosis naturally gives less striking results, but these are of value when correlated with clinical findings.

PRESIDENT DEAN: Do you expect any marked change with mural thrombi?

DR. TOBEY: At times; significant when put together with the other clinical symptoms, but I do not attempt to make a diagnosis of mural thrombosis simply by a manometer test, and I should like to impress that this manometer test is not given or even spoken of as a definite diagnostic test. It is simply a help in putting your whole picture complex together.

DR. KOPETZKY: Dr. Tobey, does the side upon which the patient is resting, in your experience, have any influence upon the differences that you obtain, pressing the right side and the left side alternatingly?

DR. TOBEY: Yes; I am going to speak of it in just a moment.

DR. KOPETZKY: Thank you.

DR. TOBEY: Let us consider some of the pitfalls of the technique as encountered in the study of a large number of cases:

1. Incomplete compression of the jugular vein due to faulty technique in applying compression accurately, as a result of obesity, tumors, cervical adenitis, or phlegmon of the neck.

2. Absence of rise in fluid pressure or very slight rise is occasionally noted in patients with no obstruction either in the venous or in the subarachnoid fluid system, when the cerebrospinal fluid is under low tension. This can easily be proved in such a case by injecting from 10 to 20 cc. of physiologic sodium chloride solution, whereupon normal dynamic tests will appear. Of course, with a mural thrombosis, or infection of the lateral sinus, your pressure is practically always positive and high. In other words, in the presence of a high fluid pressure, a reduction of the pressure to approximately 150, which is the normal, is quite necessary. Very often if you attempt a manometer test with a pressure of 300 or 350 your results are very unsatisfactory. So you should reduce that pressure to as near 150 as it is possible without causing too much discomfort to the patient, but it must be reduced. The reading above 200 is very unsatisfactory.

3. An abnormal rise in fluid pressure may result from carelessness in performing the test or from extraneous acts on the part of the patient, thus vitiating a correct record.

4. Anatomic variation in the size of the lateral sinuses, even the occasional absence of one sinus is admitted, and these facts should be considered in coming to a diagnostic conclusion.

We have made this observation, Dr. Kopetzky, which you asked about. The rise in fluid compression appears to be somewhat less when the vein on the dependent side is compressed. Does that answer your question?

DR. KOPETZKY: Yes. Therefore, it is our custom to take the reading always from the upper side and to change the position of the patient.

PRESIDENT DEAN: Dr. Hartmann, do you know of any case or cases where the anatomic variations did come into the picture?

DR. HARTMANN: Yes; we have had at least three cases where they came very much into the picture. I was going to say, Dr. Kopetzky, I wish you had said something about that position a few years ago, because that has bothered us for some years, and it wasn't until the last year or so that we sort of became sure of that fact and turned our patients around before completing the test.

DR. TOBEY: We had observed that over a period of years.

That the manometric test is of great value is shown by the fact that since the introduction of its use at the Massachusetts Eye and Ear Infirmary thirteen years ago, it has been proved correct in 80 per cent of the cases, allowing but 20 per cent for inexperience, faulty technique, and anatomical variations.

I should like at this point to ask Dr. Dean his experience. I know he has been doing some work on this test in St. Louis.

PRESIDENT DEAN: I think that I would refer the experiences we have had to Dr. Hartmann. There is just one thing that I want to mention. So many otologists have come in and said that this is a terrible thing to do with a child, to make the manometric test, that we have taken a child five years of age and had him start eating his meal before the test was begun and the whole thing has been completed without any interruption, without the slightest disturbance of the child's eating, demonstrating, I think conclusively, that if it is done right, there is very little inconvenience suffered by the child.

DR. TOBEY: What have been your results as to diagnosis?

PRESIDENT DEAN: Dr. Hartmann.

DR. HARTMANN: I think these figures are fairly accurate. When we analyzed 60 cases of lateral sinus thrombophlebitis, we found that we had performed this test in 35 only of the 60, and in 30 of the 35 the test was frankly positive and helpful. In 3 it was frankly negative, and in the remaining 2 it was doubtful.

There is only one thing that I should like to emphasize here. As I gathered from your remarks, Dr. Tobey, you are thinking

mostly of your experience with adults, and I should like to have you say something about how patient one must be, waiting for an infant or a child not to cry or hold his breath, and how occasionally one must even give an anesthetic if one is going to get reliable results.

DR. TOBEY: In the majority of our cases with children we use an anesthetic.

DR. HARTMANN: What do you use?

DR. TOBEY: Ether.

PRESIDENT DEAN: Dr. Hartmann, what do you use?

DR. HARTMANN: We haven't found it often necessary to use an anesthetic, and sometimes, in the case of an infant, a feeding quiets the baby during the test; lately we have been using one of the barbiturates, seconal, with some success.

PRESIDENT DEAN: The reason for making this test in such a small number out of the 60 cases is that these cases were gathered over a period.

DR. HARTMANN: They go back to 1915.

PRESIDENT DEAN: But more recently you do it much more frequently?

DR. HARTMANN: Now our routine includes the test as one of the steps which we think necessary, or at least highly desirable, in establishing a diagnosis.

DR. KOPETZKY: It is routine with us, but we don't use ether because that in itself raises the blood pressure, the intracranial pressure, and makes the reading harder. There is no difficulty if you select the time which you want to do it in the majority of instances. With a little rectal anesthesia we put them to sleep after eating, do the test, and everything is quiet, and they just continue to sleep and wake up in an hour or so.

DR. TOBEY: Do you use avertin?

DR. KOPETZKY: Avertin, because we want to avoid the increased intracranial pressure. That is all you need.

DR. TOBEY: What experience have you had, Dr. Lillie?

DR. LILLIE: The test is not used routinely in unilateral cases, but in the bilateral cases it has been very helpful to us.

DR. TOBEY: The test is of especial value in bilateral mastoiditis developing symptoms of otitis sepsis. For example, a child, during the course of an acute suppurative otitis media and mastoiditis, developed a relatively mild suppurative otitis of the opposite ear. Six days later, symptoms of sinus infection appeared. The manometer test indicated definite obstruction on the side of the recent involvement, where there was no evident mastoid complication. Exploratory operation on this side demonstrated complete thrombosis of the bulb and a patent sigmoid sinus. And that is where I agree with Dr. Lillie, that it is of especial value when you have a bilateral otitic sepsis to help to localize the side infected.

The most satisfactory and conclusive method of diagnosis in a suspected thrombosis is free incision of the lateral sinus.

A study of the previously enumerated symptoms, excepting the manometer test, must impress one with the fact that a careful differential diagnosis is essential. It is especially true of the case presenting explosive septic symptoms with a sudden rise of temperature, chills, leucocytosis, and possibly a bacteriemia. This often is the picture presented before an obliterating thrombosis of the sinus has formed, thus emphasizing the value of the manometer test.

In following out a series of manometer tests on a suspected case, we have found a perfectly patent lateral sinus tonight, and within twelve hours have demonstrated complete block, followed by the actual demonstration of a sinus block in the sigmoid.

PRESIDENT DEAN: It is evident that in some cases you repeat this test.

DR. TOBEY: Oh, decidedly yes, especially in a case where we can't quite make up our minds as to just exactly what is going on.

PRESIDENT DEAN: May I ask you, Dr. Hartmann, how often you repeat this test in suspected infection of the sinus?

DR. HARTMANN: Well, I know we have repeated it in a single individual at least three or four times, but I should say that we generally will repeat this test as long as we are still doubtful about the case, until we are absolutely sure that the phlebitis is there or not.

PRESIDENT DEAN: Is that in agreement with your view, Dr. Tobey?

DR. TOBEY: Yes. I think that the idea that diagnosis of a lateral sinus infection is a very simple matter is not borne out in practice.

The most frequent complication simulating the symptomatology of lateral sinus infection is a localized central pneumonia. In children especially, an area of central pneumonia may be in existence for several days, giving rise to a typical septic temperature with chills and leucocytosis, the physical signs in the chest not appearing for seventy-two to ninety-six hours. Acute septic endocarditis often shows few physical signs; the accompanying leucocytosis, however, is much higher than is encountered in sinus infection.

Acute otitis of the other ear, especially in children, may be overlooked. Acute pyelitis is not uncommon in children and, as a pyelonephritis does in the adult, may give rise to symptoms of septicemia. Erysipelas with delayed local signs, before and after a mastoid operation, has more than once occasioned a diagnosis of lateral sinus infection. Malaria, acute tonsillitis, rheumatic fever, and the intercurrence of the acute exanthemata must all be considered.

With the formation of the thrombus, the diagnosis is simplified owing to the added signs of venous stasis. At this point I might mention the fact that an early diagnosis of cerebellar or cerebral abscess is often obscured in the presence of lateral sinus thrombosis.

When the diagnosis has been made and the proper surgical procedures have been carried out, a recurrence of symptoms of sepsis indicates that there are other factors to consider. The formation of a pus pocket in the jugular bulb may need further drainage. A toxic or true metastatic focal infection may be taking place. Retrograde extension of the thrombosis toward, or even involving the other sinuses, is not unusual, and gives rise to intracranial signs of marked venous stasis.

DR. KOPETZKY: Dr. Dean, may I ask you if, in bilateral mastoiditis, you have seen a case of bilateral sinus thrombosis, and what is the method of diagnosis of bilateral thrombosis; also have none of us ever made a mistake and opened the wrong sinus while the thrombus continued on the other side, and what are the signs and symptoms that induce you to proceed immediately to rectify this perfectly human error and operate on the other side?

PRESIDENT DEAN: I have never seen a bilateral involvement, except, as you suggest, when there has been an unfortunate opening of the wrong sinus.

DR. KOPETZKY: Those symptoms ought to be stressed, too, the symptoms that the opening of the wrong side present, because they present, in the few cases that I have seen, very definite symptoms.

PRESIDENT DEAN: Tell us about the symptoms.

DR. KOPETZKY: There is an enlargement, a general edema of the head, the eyes sink in, the whole head becomes edematous. That lasts for about three to five days and then gradually subsides. I have seen this where the wrong sinus has been opened; and everybody viewing this enlargement of the head, becoming very panicky, brought the case in for treatment, and I have seen, where a bilateral diagnosis was made, and since a fatal outcome was expected, surgery was done on both sides. The same edema develops. Since collateral circulation must eventually be established to take down that edema, everything possible must be done to sustain that patient in the interim by blood transfusions. Then, if possible, have an interval of time elapse between the first operation of the first side and the operation of the second side. In that way I have handled the very few such cases that have come to us. It is remarkable that in the literature on sinus thrombosis, and the traps that you meet in handling it, this particular trap is never talked about, and the literature is strangely silent on this bilateral lesion, and on a record of wrongly diagnosed sides upon which operation has been undertaken. Yet it must happen!

PRESIDENT DEAN: How long do you think it would take to establish collateral circulation?

DR. KOPETZKY: Judging by the rapidity with which I have observed this edematous condition of the head subside, I should say within three or four days, and it is usually gone at the end of a week.

DR. TOBEY: Do you control this increased cerebral spinal fluid pressure by repeated lumbar punctures during this interval?

DR. KOPETZKY: Both by repeated lumbar punctures and by draining some blood out, leaving the body with less blood bulk, but the second procedure is rarely necessary, nor is the first necessary in most instances. The proper thing to do is to put the patient in a half upright position and help him by gravity to restore the hemoglobin which is being destroyed by transfusion, and operate on the second side as soon as you think it is reasonably safe. After all, you are faced with a very desperate situation.

PRESIDENT DEAN: We have intentionally prolonged the discussion of the diagnosis because the diagnosis is the important thing. Without the diagnosis you can make no progress in the handling of the patient. We will go ahead more rapidly with the other contributions. Dr. Furstenberg.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

* * *

A. The Routes of Infection of the Sinus

B. The Anatomical Variations of the Sinus and the Effects of These Variations on Diagnosis and Treatment

A. C. FURSTENBERG, M.D.

ANN ARBOR, MICH.

ANOMALIES of the circulatory system are frequently observed, particularly those which involve the venous system. It is probably true that the sinuses of the dura mater rarely show anomalous development because they are found more or less constant in studies of many skulls in the anatomical laboratory and in the numerous clinical experiences that bring them conspicuously before the otolaryngologist. Their occurrence must be exceedingly infrequent because in the lifetime of otologists of wide experience they are observed in only rare instances during operations upon the temporal bone. However, anomalies of the sinuses do occur, and when they arise and are the seat of infection, they cause considerable confusion in diagnosis and methods of treatment.

It is my purpose at this time to enumerate and describe the various anomalies with which I have had personal experience either in the operating room or in the anatomical laboratory and discuss a surgical approach to them when operative interference is demanded. A discussion of this character which involves surgical technique in the treatment of infected anomalous lateral sinuses must, in a large measure, constitute theory and surgical speculation. Too few cases are seen in the lifetime of any otologist to permit him to standardize technical procedures suitable to the various anomalies and the innumerable clinical factors and influences asso-

ciated with them when they become the seat of an inflammatory process. Theory and hypothesis can be reduced to scientific facts only after long clinical experience and experimentation of a practical nature. I lay no claim, therefore, to originality in this work or to the development and standardization of any acceptable surgical technique for the treatment of diseased anomalous sinuses. I merely present the various abnormalities well known to all of us, relate my own clinical experiences with those that I have encountered personally, and theorize on procedures that might be employed for those which have never come to my attention.

I often tell my staff members and medical students that whenever I discover a subject in otolaryngology about which I know nothing, I give a course in it, and that is my status at the present time. Reputations are often made on this basis. I recall the irascible pedagogue who taught my senior class tropical medicine. He had never traveled south of Toledo, nevertheless he became an internist of considerable repute.

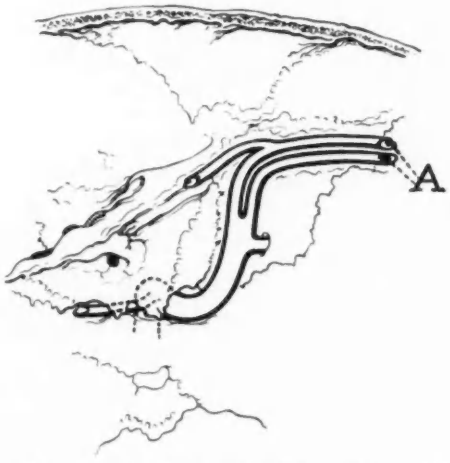


FIG. 1 — (Furstenberg) — Double lateral sinus (intracranial view).
A. Two lateral sinus.

Your attention is called to that rare anomaly of the lateral sinus in which a horizontal lacuna has divided the transverse or sigmoid portion into two separate channels (Fig. 1). You will readily note that in searching for the infected one, the normal sinus may be opened and the case labeled one of mistaken diagnosis. Then, too, a thrombus may be found within one division of such a sinus, the process dealt with adequately and the second division, which is

perhaps likewise affected, may be entirely overlooked. Such an anomaly offers an exceedingly perplexing problem when dealing with lateral sinus thrombosis. The only warning which can be sounded is to keep in mind this anomalous possibility and scrutinize carefully the normal pathway of the lateral sinus for the possible existence of a divided structure when dealing with a sinus thrombosis.

FIG. 2 — (Furstenberg) — Absent lateral sinus. The sigmoid takes its origin from a large superior petrosal sinus (intracranial view).
A. Large superior petrosal sinus. *B*. Absent lateral sinus.

A second anomaly of rare occurrence is that in which the transverse sinus is absent and the sigmoid takes its origin from a large superior petrosal sinus (Fig. 2). Such an abnormality causes great confusion when, after removing the septic thrombus and securing free hemorrhage from above, you encounter great difficulty in controlling the hemorrhage. You are amazed to discover that the usual pressure applied between the skull and the region of the lateral sinus fails to control the hemorrhage. In one such case that has come to my attention, I was unable to control the hemorrhage with a pack under the tegmen of the mastoid at the knee of the sigmoid sinus in the region of the superior petrosal sinus, presumably because the latter structure was exceedingly large and did not lend itself to compression by this method. Only after considerable difficulty was I able to control the hemorrhage by opening the sigmoid sinus widely and packing vigorously into the lumen of the large superior petrosal sinus.

It is not uncommon to find a small contracted jugular foramen with a small bulb and jugular vein and the main volume of blood passing through a much enlarged mastoid emissary vein (Fig. 3). Because of the enlarged emissary vein, this structure is exposed to trauma during the mastoid operation and hemorrhage from it may be controlled with difficulty. Likewise, in such cases, a thrombosis of the sigmoid sinus produces a similar pathological change in the emissary vein which sometimes approaches the proportions of the main vessel and requires considerable surgical effort in causing its ablation.

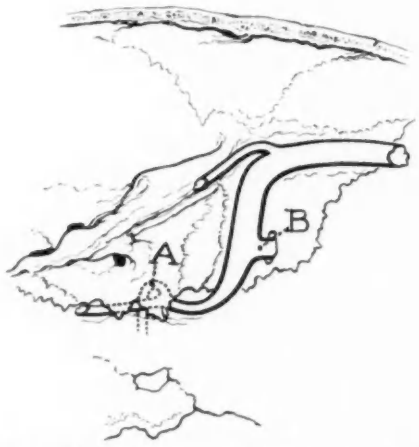


FIG. 3 — (Furstenberg) — Constricted jugular bulb and distal end of sigmoid sinus, the sinus passing through a small jugular foramen (intracranial view).
A. Small jugular bulb. *B*. Large emissary vein.

There is another type of anomaly in which the lower portion of the sigmoid sinus is absent and the latter structure ends in a blind pouch with no direct communication with the internal jugular vein (Fig. 4). The flow of blood through such a sinus is largely through an enormous emissary vein, but in some instances also through a persistent petro-squamosal sinus. The mammalia in general, below the anthropoid apes, present only a very small internal jugular vein, which arises as an ascending collateral branch of the external jugular. It merely reaches the base of the skull and receives little or no blood from the intracranial structures. In this anomaly the lateral sinus does not turn downward to form the sigmoid portion but continues forward along the petro-squamosal suture in a bony groove over the superior surface of the petrosa and

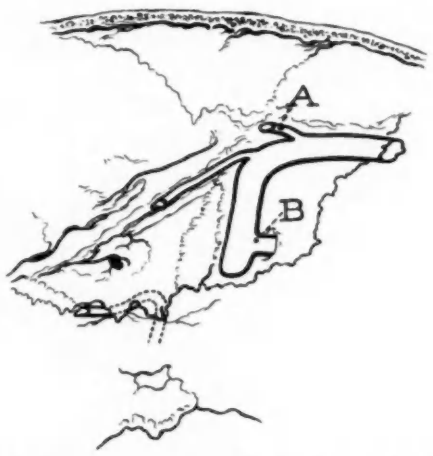


FIG. 4 — (Furstenberg) — Sigmoid sinus ending in a blind pouch. Circulation is conducted through a patent petro-squamosal sinus and a large emissary vein (introcranial view).
A. Patent petro-squamosal sinus. *B*. Large emissary vein.

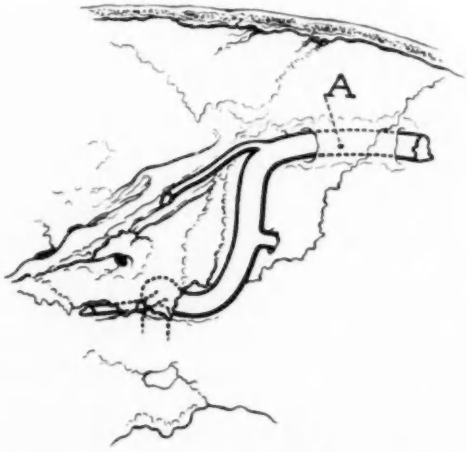


FIG. 5 — (Furstenberg) — Portion of lateral sinus occupying a position external to the skull. It lies immediately beneath the pericranium over the temporal and occipital bones (intracranial view).
A. Lateral sinus external to skull.

leaves the skull by the temporal foramen in the squamous portion of the temporal bone. Thus, the petro-squamosal sinus reaches the exterior of the calvarium and the external jugular vein between the external auditory canal and the temporo-maxillary junction. It is obvious that when dealing with this type of anomaly, ligation of the internal jugular vein would be of no avail. Not only is it necessary to remove the thrombus from the sigmoid sinus or drain that structure, but similar care must be given the large mastoid emissary vein and the vestigial petro-squamosal sinus. I have seen one case in which a septic thrombus was traced for a considerable distance and removed from a persistent petro-squamosal sinus of large proportions.

Rarely does one find a portion of the lateral sinus, and sometimes, too, a part of the sigmoid sinus, lateral to the skull in the soft tissue of the scalp (Fig. 5). I have never encountered such a case in clinical experience, although I was called to the anatomical laboratory several years ago to see a specimen in one of the cadavers. Obviously, such an anomaly leaves the lateral sinus widely exposed to the surgeon's knife when the primary incision for the mastoid operation is made. Several anomalies of this character have been reported in the literature, although little difficulty seems to have been encountered in controlling hemorrhage produced by trauma of such an anomalous vessel.

I have seen two specimens on the operating table in which the transverse and sigmoid portions of the lateral sinus contained lakes (Fig. 6). They represented irregular, dilated portions of the vessel and when opened caused great apprehension about our ability to control the hemorrhage. Only once in my experience have I encountered hemorrhage from the lateral sinus that seemed for a time to be beyond my control and this was in a patient who presented the anomaly above described. Because of the presence of an acute suppurative otitis media and a septicemia, sigmoid sinus thrombosis was suspected. The vein was exposed and opened, but no thrombus found. The bleeding which ensued continued unabated for nearly two hours and taxed every resource of Dr. Canfield and myself to bring it under control. Confusion reigned, because with the removal of each portion of the inner table of the skull in an effort to make a wider exposure of the transverse sinus so that adequate pressure could be applied, more lakes were opened and new sources of hemorrhage created.

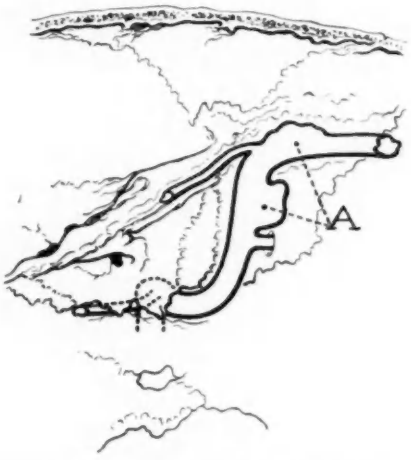


FIG. 6 — (Furstenberg) — Lateral sinus showing two large lakes (intracranial view). *A*. Lakes.

Lastly, one may encounter a large petro-squamosal sinus which carries considerable blood from the lateral sinus to the veins of the soft tissues in front and below the temporal region (Fig. 7). If one does not recognize the possibility of such an anomaly, considerable confusion arises in attempting to identify it when it is observed springing from the region of the knee of the sigmoid sinus. In one instance, I have seen it the seat of a septic thrombus.

Let us consider the methods of spread of infection responsible for the production of a sigmoid sinus thrombosis. Perhaps the most common pathway of extension is by continuity of tissue. The cells of the mastoid undergo dissolution, the osseous sigmoid plate is destroyed with direct exposure of the sigmoid sinus and the production of a peri-sinuous abscess. From this lesion there follows an injury to the walls of the sigmoid sinus and the pathological changes responsible for the production of a septic thrombus.

It is likewise obvious that a thrombus of the sigmoid may arise as the result of infection extending through the efferent diploic veins directly into the lumen of the lateral sinus. Within the small diploic veins a septic thrombus develops which by antegrade extension eventually involves the sigmoid sinus: the structure into which these veins empty.

Your attention, however, is called to another method of spread which may be rather rare but nevertheless common enough to be

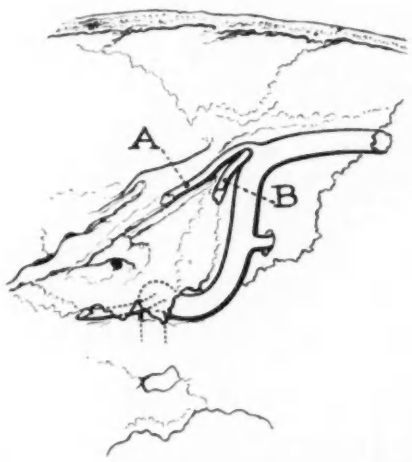


FIG. 7 — (Furstenberg) — Persistent petro-squamosal sinus (intracranial view). *A*. Superior petrosal sinus. *B*. Petro-squamosal sinus.

regarded as a clinical entity. I refer to that process in which there is produced, as the result of extension of infection through the efferent diploic veins, a septic exudate in the potential space between the sigmoid plate and the dura mater. Such an exudate of varying degrees of organization may remain beneath the osseous sigmoid plate for days, weeks, and even months without destroying it. A septic thrombus within the sigmoid follows and the disease may run its course even to death without destruction of the sigmoid plate. It is this type of spread which offers an exceedingly perplexing problem when, at the time of the mastoid operation, an effort to diagnose sigmoid sinus thrombosis is made. The surgeon discovers what appears to be a normal sigmoid plate and fails to realize that an exudate of great virulence and one which is a potential danger to the sigmoid sinus may lie hidden beneath it. An inflammatory process of this character and in this region may lie dormant for a period of weeks or even months without gross evidence of injury to the osseous plate of the sigmoid.

PRESIDENT DEAN: Dr. Kopetzky.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

* * *

A. The Pathology. *B.* The Bacteriology. *C.* Immunological Treatment

SAMUEL J. KOPETZKY, M.D.

NEW YORK

YOU HAVE given me three points, Dr. Dean, the pathology, the bacteriology, and the immunological treatment. Some of the pathology has been touched upon. To understand the pathology, first one must know what a thrombus is and how it comes about. A thrombus is a defensive mechanism of the body surrounding an infection that gets into the blood vessels. The factors involved are *an injury to the intima, infection and trauma,* surgical or otherwise — and that is why we objected to the needle puncture in an effort at diagnosis, to the intima — *slowing of the blood stream,* and then the fact that the thrombus grows about the site of an infection and trauma, and grows both with and against the blood current. Next, thrombi and phlebitis develop in two categories, and I, therefore, find myself happily in accord with Dr. Lillie, who thinks that we should individualize on our cases in determining on therapy, and one should individualize on them, also, when one is making determinations for diagnosis.

In one group, the sinus is reached by the breakdown of contiguous tissue. In the other, Dr. Furstenberg has already outlined the route by which through the efferent veins the interior of the sinus is reached while its outside wall remains unaffected. In the first group, where the sinus is reached by contiguous tissue, one will expect to find perisinal abscess and granulations on the outside of the sinus. Where one expects the normal-looking vein wall, one must take into consideration that the lesion had not reached

the sinus by the contiguous infection of the tissues, but has reached its interior through the development of thrombi which eventually coalesced, brought deposits of thrombus fibrin together in the vein's interior, and created a thrombus in the larger vessels with a normal-looking external wall.

With this in view and to hurry the time, I want to show a set of lantern slides to trace a thrombosis from the subepithelial tissue in a mastoid cell to the vein. It is not so important to study microscopically the advance of a sinus thrombosis which develops by the contiguous route, because such a lesion is very obvious at the operating table, but the other type is the one more often overlooked, not diagnosed or misdiagnosed, or the operation on it is too often delayed. (Demonstrates slides).

I present a slide showing a mastoid cell. I show you the lining membrane of the cell, and in the subepithelial layer there are these dark spots which are thrombi in the walls of the lining membrane of a mastoid cell.

A further advancement of the lesion is seen in the membrane lining of the mastoid cell, which is now much engorged. One sees empty veins, but also some with living streptococci in them. These slides are some from the studies that one of my associates, Dr. Julius Bell, is making on streptococcic infection of the mastoid.

The next slide demonstrates a thrombus in the veins, running in a haversian canal in the beginning stages of an infection in an aseptic case, while the next slide shows an enlargement showing living streptococcus hemolyticus in those veins of a haversian system seen with higher powers of magnification.

Here is a streptococcic piece of bone with a haversian system, streptococci in it and the thrombus in the veins, with an enlargement of that thrombus and that system, observed under higher power.

Then I throw a picture on the screen of the hypotympanum. I demonstrate connecting veins. I demonstrate the thrombosed jugular bulb, and point out in tissue spaces in the place that Dr. Furstenberg just diagrammatically showed you, there are distinct round-cell infiltrates. The slide is from Witmaack's.

I show another one from my own specimens. It shows hemorrhage mastoiditis taken from a case of influenzal mastoiditis with real influenza. Note the intact bony walls. Look at this vein with a thrombus in it. We proved the deposit by staining it with the Weigert stain, which identifies fibrous tissue and is a selective

stain. Then we demonstrate the many thromboses of the vessels in a mastoid process whose cells were not coalesced.

These cases all give septic symptoms from the start, but there is no breakdown in the cells, and the finding must not be confused with a commencing coalescing process; distinctly, this is not a beginning coalescent mastoiditis. It never will become coalescent It is an entirely different lesion.

I show you an enlarged picture of that vein, which was studied at various stages to show the fineness of the deposit in it, whereas other veins are shown to be empty.

In contrast to what I have already shown you, I now show you empty veins with no thrombus in them but red blood cells, as a control picture.

Now look at this jugular vein, three days after the onset of the lesion just demonstrated; a normal portion of the vein showing the intima of the jugular, some enlargement of the jugular vein, and the blood plasma area are quite clear.

Now the blood plasma area has disappeared. Hemolysis is showing in the vein, and a beginning hypertrophy and infiltration of the vein wall become evident.

The vein wall becomes exceedingly thick, fluid blood here, and now observe the thrombus in the tissue of the vein's interior.

The jugular vein is here so swollen that it turns upon and folds upon itself. This is hemolysis and thrombus in a vein a few days after the onset of an acute hemorrhagic mastoiditis, with septic symptoms.

Finally, I demonstrate a slide which is a picture of a vein that was not resected and not ligated. The lateral sinus alone was handled. Subsequently, sepsis again developed, and other portions of the specimen showed definite recanalization of the unoperated vein, but in its walls, secondary vessels became infected and thrombosed, and then because of the clinical sepsis, the jugular vein was resected. The patient recovered and this specimen resulted, which substantiated our own particular viewpoint that in all these cases safety would indicate that the jugular vein should be resected.

Under these circumstances, then, with that picture and with what has been presented here before on diagnosis, bacteriology plays a rather simple part, because in the large majority of infections with which I have had to deal we have met only one organism, namely, the streptococcus hemolyticus pyogenous. Occasion-

ally, we have a sepsis and the bacteria gives us a pneumococcus. In such we do not touch the lateral sinus, because we do not believe a pneumococcus creates the thrombosis. So the bacteriology resolves itself to the identification, if possible, of a streptococcus hemolyticus in the blood stream, or in the tissues, and I think in a recent issue of the Archives we have had published from my staff a method of growing these bacteria in tissues. The study is possible from spicules removed at operation. So that one can know for sure whether or not he is dealing with a streptococcus hemolyticus.

Immunological treatment I have not found of much avail. Blood transfusion to replace destroyed blood tissue elements that a hemolytic infection removes from the body is logical and we do it; namely, we give whole blood in small transfusions. If we have identified the organism in the blood or otherwise, and a sepsis is persisting, and we need a secondary help in the treatment, we immunize a matched donor. In the majority of cases, however, adequate surgery is successful so that the matched donor is not needed because the case has resolved itself before the time the donor would be ready. In the absence of a matched donor, postscarlatinal blood is of great advantage. That is as far as we have gone in immunological treatment.

We started chemotherapy last year in conjunction with surgery, and in conjunction with our other supporting measures. It is not yet clear to us what real value to place upon it.

DR. LILLIE: May I ask a question, Dr. Dean? I should like to ask Dr. Kopetzky if I understood him correctly to say that he ligates the jugular vein in all these cases.

DR. KOPETZKY: No, sir.

DR. LILLIE: What makes your choice of ligating the jugular vein or not?

DR. KOPETZKY: My position is very similar to yours, sir. Given a sepsis and the other supporting signs of a thrombosis, I do not go into the neck first at all. I go into my mastoid, where the obvious lesion is located. I look at that vein. If the surface is diseased, and it gives me reason to go into it, I will go into it. I open it, remove the clot, and plug it. Then if it has been thoroughly thrombosed and the circulation completely shut off, I will rest. If the throm-

bosis shows no signs of having broken down or of having become distinguished from the rest by discoloring, as it ought to be in the early stage, I will rest. If the thrombus is beginning to show disorganization or breakdown, I don't dare rest, because pieces may go off into the circulation. On the other hand, finding a mural clot, where I have no means macroscopically of estimating how far that vein wall is involved, I do not rest. I continue surgery on the jugular. If I have rested, I expect a prompt remission of septic symptoms. If I do not get them, I do not want autopsy material. I immediately ligate, resecting my jugular first. I get the specimen and the patient continues on!

DR. TOBEY: Do you follow out the same procedure when you have a jugular bulb thrombosis where it is impossible to get free bleeding?

DR. KOPETZKY: If I have a jugular bulb thrombosis, or a primary bulb thrombosis, I go into the neck. I must, logically.

PRESIDENT DEAN: Dr. Lillie, does that meet with your approval?

DR. LILLIE: I like to individualize my patients. I like to get the case of sepsis at the earliest possible moment so that I have the least to do on it, and I depend greatly upon his general state of health as to how far I will go at the primary operation. If my patient is severely ill, I, too, immediately go to the neck and then into the mastoid. If he is in a relatively good condition, I prefer to go into the mastoid. There, if I find a hemorrhagic mastoiditis, even though the sinus plate appears normal, I remove it. Frequently I have found, in removing the sinus plate, it is found to be adherent to the sinus surface and little petechial bleeding points are present. If the patient is in the early stage, in pretty good condition, I stop there. If the patient does not do well from there on, then I have access to other surgical manipulations and procedures without having jeopardized his chances for recovery.

DR. KOPETZKY: I might give another diagnostic sign. When you come down and expose a lateral sinus and the vein wall looks normal, if it pulsates, it is thrombosed. If it does not pulsate, it *may* not be thrombosed.

DR. LILLIE: I think we are all talking about several different things. There are several stages of the disease, the early stage, the middle stage, and the end stage. If we encounter the sepsis early in the disease we have to do less. If we encounter sepsis in the middle stage of the disease, where a thrombus is forming in the sigmoid sinus, we have to do more. If we encounter sepsis in the end stage, where all the mischief has taken place, then we must adequately remove the primary focus and in addition must attempt to close avenues of further extension to the blood stream. It is extremely important that the pathologic conditions causing sepsis be thoroughly understood if stages of the disease are to be recognized. Otologists should talk on common ground. One should not be talking about thrombophlebitis as it occurs in hemorrhagic mastoiditis and another talking about septic thrombosis as it occurs in suppurative mastoiditis, if we are to discuss adequately the therapy. That is what Dr. Kopetzky implies.

DR. KOPETZKY: We are agreed.

PRESIDENT DEAN: The question of blood transfusion and hemotherapy, and the surgery in the neck will be more thoroughly discussed when we have the last two contributions. In short, I think things have been brought up by Dr. Furstenberg and Dr. Kopetzky, and the things that Dr. Lillie is going to mention, will, most of them, be discussed when we come to the question of medical and surgical treatment which is secondary to diagnosis.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

Papilloedema, Brain Abscess, and Meningitis Complicating a Septic Thrombophlebitis of the Sigmoid Sinus

H. I. Lillie, M.D.

ROCHESTER, MINN.

In the discussion of certain of the complications of septic involvement of the blood stream associated with mastoid disease, it seems important that the underlying physiologic and pathologic fundamental aspects be reviewed. Full realization of the importance of the phenomena makes easier the understanding and explanation of the occurrence of the manifestations of intracranial extension. Reduction of the occurrence of certain complications to statistical percentage is fraught with great danger because the case in hand must be dealt with as a distinct individual problem.

The extensions of infection into the intracranial structures are largely direct. I think it is important that the cerebrospinal fluid be understood. It originates by secretion from the choroid plexus in the ventricles, it circulates slowly through the ventricles, through the tentorial notch, goes out over the cortex, and is absorbed in the arachnoid villi, in the pacchionian bodies and nerve sheaths.

The blood vessels pass from base to substance, supply nuclei and central white matter; reaching the region of the ventricle, they turn outward, and terminate as end arteries.

The cortical vessels pass around over the surface in the subarachnoid space. The small ones pass into the substance of the brain, and the larger ones into the sulci.

Between these two is the avascular zone. This area is very important, because abscesses usually originate in this zone.

The veins empty into the blood sinuses and the vertebral veins. The venous system is a very complex arrangement. Because of this anatomical complexity of the venous system, recently there has arisen the pronouncement from anatomists that the time-honored and successful surgical attack on septic thrombosis is misdirected and futile. From an academic standpoint, this may be so, but the practical experience of competent observers for the past half century would seem clearly to refute the contention.

Any interference with the circulation of the blood or of the circulation of the cerebrospinal fluid produces mechanical effects.

Increased pressure is due to some disturbance within the absorbing mechanism of the spinal fluid. There may be no pathological changes in the choroid plexus, the secretory mechanism, but in meningitis there may be irritation or interference with the absorbing mechanism as the result of toxins or bacteria on the pacchionian bodies and villi. This has been shown pathologically and microscopically to consist of débris and broken-off cells.

If the veins or sinuses are obstructed by thrombus or occluded by pressure, the effect is marked because the collateral circulation in the brain is unlike other parts of the body. Retrograde thrombosis may be protective in one sense, as is claimed, but may be the cause of this increased intracranial pressure. Involvement of the straight sinus increases the intracranial pressure greatly.

The anatomical arrangement of the torcular herophili has considerable effect upon intracranial pressure. It is known from anatomical studies that in 50 per cent of the cases the longitudinal sinus empties into the right lateral sinus, and in 50 per cent the straight sinus empties into the left lateral sinus. In 30 per cent the torcular herophili may be confluent or it may be a cistern-like thing, with portions of the load going in any direction, and, therefore, it has great possibilities when it is obstructed from thrombosis.

Choked discs occur in the course of mastoid disease and merely show the effect of increased intracranial pressure. In my experience in sinus thrombosis it is more likely to have occurred after my operative measures upon the sigmoid sinus or jugular vein. I explain this on the basis of back pressure. It is like damming up a stream, and the compensatory circulation, not having been established, naturally it results in a purely mechanical failure. The significance must be determined by repeated examinations.

It must also be understood that there can take place as a result of this back pressure the choking of other nerves. I have seen what

I thought to be a choked eighth nerve, and the loss of function of the vestibular branch and the auditory branch. Sometimes it is difficult to differentiate between the effect of a neuritis in the eighth nerve, labyrinthitis, or choking.

I think, also, this occurs in the olfactory nerve, and the fifth, sixth, seventh, eighth, ninth, and tenth, as we see in the jugular foramen syndrome. It is known that the circulation in the brain is that of a closed box, the arterial blood coming through the arteries, fill the brain on a certain phase of respiration, and the sinuses take up the pulsation, so that there is no throbbing. When that is interfered with too much it produces throbbing headache. That is the explanation for the pulsation of a thrombosed sinus that Dr. Kopetzky called attention to. There is no give to the walls of the sinus and, therefore, it pulsates with the intracranial arterial pressure impulse.

Increased pressure may be inflammatory in origin. It may come as a result of inflammation of the meninges, and if increased to a certain stage it produces certain signs and symptoms with which we are greatly concerned. I should like to list these under so-called otitic hydrocephalus, which is probably a severe aseptic meningitis, pseudo brain abscess and abscess. Acute hydrocephalus, I think, usually comes on rather suddenly and is usually present in younger patients, children particularly. It is initiated by severe headache and followed by various degrees of stupor, and the general picture of serious illness. Particularly conspicuous in this type of situation is the choking of the optic disc. The diagnosis of otitic hydrocephalus can be verified by tapping the spinal fluid and finding marked increase in pressure of the spinal fluid. If spinal drainage does not relieve the syndrome relatively early, then one may consider tapping the ventricle. Tapping the ventricle is a relatively easy operation, or at least is easily performed. The use of dehydration measures earlier in the course may be more effective.

Dr. Kopetzky: Where you suspect Symond's hydrocephalus, or otitic hydrocephalus, as you call it, the diagnosis is very definitely made by tapping the ventricle, putting oxygen in it and taking an x-ray picture. You can then see it, and it definitely rules out a brain abscess because of the symmetry that the ventricles show to you on the picture.

Dr. Lillie: But the tapping of the ventricle is so striking in this type of case, it is really dramatic.

In the acute hydrocephalus, palsy of the sixth nerve may be present. Cushing has shown the reason for that. Occasionally the sixth nerve lies under the inferior cerebellar artery and sometimes over. If it lies under the artery, increased pressure has more effect.

Pseudo abscess is, I think, a pretty fair term, or you could call it a localized encephalitis. Here the symptoms are quite similar to that of otitic hydrocephalus, except that they have even more localizing signs, and it is usually associated with disease in the adjacent structures of the temporal bone, in either the cerebrum or the cerebellum. Abscess in the presence of septic thrombosis of the sigmoid sinus would inferentially be more likely to be in the cerebellum. It can occur in the hemorrhagic type of disease because of retrograde thrombosis, or it can occur in the confluent type of disease as a result of contiguous destruction of tissue adjacent to the cerebellum. It may extend through the posterior wall of the sinus.

Suppuration of the brain may occur as a localized subdural abscess, the integrity of the dura having been broken down slowly, the arachnoid sealed off, and the reparative process makes a protective wall or capsule.

If an abscess forms in the cerebellum, the neurologic signs of diagnosis are those of incoordination, etc. I do not think that this is the place to go into diagnosis minutely. It is possible that abscess or localized encephalitis may occur in the cerebrum from extension along the superior petrosal sinus, and it can at least be said that the diagnosis in the acute stage is relatively difficult.

In general, then, it can be said that the signs and symptoms referable to intracranial extension are those of pressure and localized lesions. The general signs are always vague. They are general malaise, particularly headache; mental changes; stupor, the patient will answer direct questions but he is not particularly interested in his surroundings; vomiting is an inconstant sign; temperature and pulse are usually high at first because of the presence of the acute infection. If one waits until the classical signs are present, then it might just as well be a brain tumor as a brain abscess. I mean the lowering of temperature and the lowering of pulse, which are late signs. Convulsions are frequently present, particularly in children. Why they should be more prevalent in children than adults, I do not know, but they are signs of some irritation, particularly of the temporal lobe.

It has been variously estimated that choked discs are present in about one-third of the sinus thrombosis patients who are exam-

ined, but vascular changes showing dilatation are more common than is the choking of the discs. The visual fields may change, and they are of the same significance as they would be in brain tumors. Therefore, it is said that the signs of involvement of the cerebellar structure are those of incoordination, loss of muscle tone, ataxia, dysmetria, tremor, speech slurring, gait, etc.

I do not know what to say about meningitis. We know how it occurs. We are always very much concerned. If, however, the extension of infection to the brain is recognized at its earliest stage, considerable benefit may be expected by removing the focus from which the meninges may have been involved. The meninges are involved by continuity or by retrogression of thrombi back through the meninges into the arachnoid space, and the infection is of such a fulminating character that the reparative process is not of sufficient force to cause a localization.

The use of spinal puncture in these suspected intracranial complications gives much information. If the symptoms are due to increased pressure it may relieve the syndrome dramatically. If on microscopic examination, evidence is found from the cell count and the chemical examination that infection is present, then we are on our guard. If we find evidence of bacterial involvement of the meninges in the early stage, we should proceed with the removal of the original focus; although the prognosis is bad, there are a sufficient number of cases reported where successful management has taken place to warrant surgical interference. If it is found to be definitely suppurative meningitis, then I think that any surgical interference is misdirected.

PRESIDENT DEAN: We will not discuss the question of meningitis, pseudo brain abscess, and brain abscess, because of time allowance. It is necessary, however, to mention these things briefly, as Dr. Lillie has done, in order to thoroughly understand the subject under discussion. We now have fifteen minutes to consider medical and surgical treatment, which are second in importance only to the diagnosis, and I trust that from now on the discussion across the table will be active.

Dr. Hartmann.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

* * *

Medical Treatment

ALEXIS F. HARTMANN, M.D.
BY INVITATION
ST. LOUIS

WHEN I sat down here, Dr. Kopetzky wanted to know how a pediatrician ever got with this group, anyhow, and Dr. Lillie suggested that it was probably because the subject was child's play. Sometimes I feel like a fifth wheel, but on the other hand, sometimes all we have to do is to make a diagnosis and get the patient in shape so that he can stand an operation and keep him alive long enough to get over it. So maybe we have some place in this conference.

Now, we want to look at it broadly. The medical treatment of lateral sinus thrombosis should be no different from the medical treatment of any other severe infection, and we should carefully study changes that go on in the body in the course of very severe infection. We know something of such changes. Sometimes we can correct them and prevent them; sometimes we can't.

Now if we may have this first slide. I have here what I think are perhaps the four most important points of attack (Table I). We can have, especially in infants and children, such severe disturbances in water balance and acid base balance that of themselves these secondary symptoms of infection can determine the outcome unfavorably. We also may have another change of metabolism involving particularly carbohydrate metabolism, which permits carbohydrate stores to become depleted, which permits ketosis to occur, and which may even permit very severe hypoglycemia to develop, which in turn may interfere with efficient circulation of the blood. Then we may have direct effects on blood circulation, simply due to changes in plasma volume brought about by loss of water

from the plasma or loss of protein from the plasma, and we can have ineffectual circulation because of loss of red cells from anemia. Then again we can see patients who have such a virulent or overwhelming infection that they die long before anything surgically can be done about them.

First, I am just going to outline for you the high spots here. Later I hope some questions may be asked about details of management.

TABLE I — *Medical Treatment of Lateral Sinus Phlebitis*

I. Preserve Normal Metabolism:
   A. Water and Acid-Base Balance
   B. Blood Sugar Level and Glycogen Stores

II. Preserve Adequate Blood Circulation:
   A. Plasma Volume
   B. Red Blood Cell Volume

III. Confer Immune Bodies Passively

IV. Decrease Extent or Virulence of Infection:
   A. Drainage of Metastatic Foci
   B. Chemotherapy

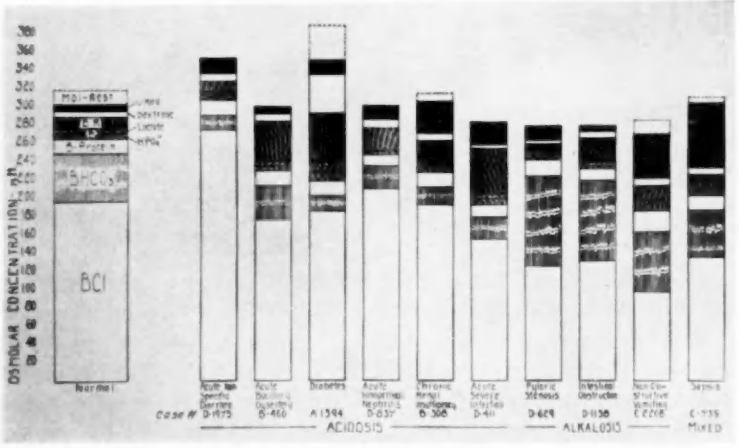


Fig. 1 — (Hartmann)

Figure 1 shows some chemical pictures or patterns of blood serum which reflect changes throughout the body water, and we have two that have a bearing in this connection. One type is fairly characteristic of acute severe infection. Now, regardless of the type of infection, we are likely to see, in infants and children, dehydration, fluid loss, with bicarbonate reduction and varying degrees of retention of waste products. If there also happens to be some kidney damage, as there was in this child, with hemolytic streptococcic mastoiditis and septicemia and acute hemorrhagic nephritis, these chemical changes will become pronounced, and include great reduction in chloride, N.P.N. of over 200 milligrams per 100 c.c., and phosphate retention of 24 milligrams inorganic phosphorus per 100 c.c. Now you have to admit that if such changes occur, they could greatly influence the general picture.

In brief, if one has any kidney function at all worth speaking of, one can prevent the water changes and the electrolyte changes by the proper administration of fluids, but by fluids we do not mean simply normal saline solution or dextrose solution. For instance, a severe type of renal acidosis cannot be relieved by dextrose solution or saline solution, because it is not brought about by factors which these substances are capable of influencing very much. One may have to give, and usually does have to give, alkali which is generated independently of any renal activity, and which takes care of an acidosis that is not due largely to ketosis.

Our own practice has been to supply in sufficient quantities such substances as sodium lactate, which can develop bicarbonate after metabolism, and Ringer's solution, which offers the body at least all the building stones necessary in the form of water, sodium, potassium, magnesium, calcium chloride, and alkali, independently of renal activity, which it may need in its recovery. Now, someone may later want to know just exactly how we can do that, and perhaps we can say something more about that later.

You can imagine when, at midnight, a child of three or four has a temperature of 106 or 107, and then within four to six hours has a temperature of 96 or 97, and has become cold and almost pulseless, that there is something really wrong with the circulation at such a time. It may be due to nothing but the infection, but it may also be due to something that you have overlooked and can correct. For instance, all the factors that operate in these various severe infections that become prolonged are factors which might lead to depletion of carbohydrate stores and permit very low blood

sugars to result and sometimes a circulatory collapse is on such a basis. For instance, we have a much diminished food intake of all types. We have a reduction in carbohydrate stores in the liver. We have a greatly increased oxidation in the tissues, with consumption of carbohydrates at a faster rate, and we may have secondary adrenal inefficiency, which decreases the ability of the liver to give up the glycogen to come to the support of the falling blood sugar. So it behooves us as we follow these patients to pay attention to the blood sugar level, or at least to make certain that the carbohydrate intake will not permit this type of hypoglycemia to occur.

You may say that can be easily done if we simply give daily, or twice daily, some intravenous dextrose to a patient who isn't very well, but I say it can't be done that simply. In the first place, there may not be enough sugar given that way, and in the second place the practice may lead to hypoglycemia. The effect of dextrose administration sometimes is only a transient rise in blood sugar to be followed by a severe dip later.

Here is an infant who has had severe dysentery. The dysentery had practically been recovered from. Instead of losing a lot of water and food by way of the intestinal tract, absorption was occurring and led to edema, which you can easily see; a very marked case of so-called nutritional edema, showing edema particularly of the extremities and the face, without much of the body. This child also had otitis media and mastoiditis due to a hemolytic streptococcus, and from this time on the mastoid picture was the important one.

At this time this child's circulation was quite inadequate and one could not demonstrate any blood pressure in the upper extremity nor even feel the radial pulse, due to the low plasma volume made possible by the loss of plasma protein due to prolonged starvation. One may find acute anhydremia with circulatory failure in the presence even of edema, simply brought about because the level of plasma protein is not sufficient to bring water back from the tissue into the circulation, and to correct a change like this will require the transfusion of whole blood, with sufficient plasma, or of plasma itself repeated often enough, or the transfusion of some substitute for plasma, such as acacia.

I am going to skip the third type of treatment, the attempt to confer immune bodies passively, because, just as Dr. Kopetzky mentioned, we are disappointed in the results with blood transfusions from immunized donors, or donors especially selected for

one reason or another, although we admit that not infrequently after transfusions of whole blood, changes occur that are so striking that we really feel that we have conferred passive immunity to such patients in that way.

We will, however, spend a little time on what little we know at the present time about what the drug sulphanilamide may do in this connection. In the early summer we had two children, one three years old and the other two years old, in whom mastoiditis was present, very difficult to diagnose, and in whom persistent septicemia was noted, and on whom operation proved lateral sinus phlebitis.

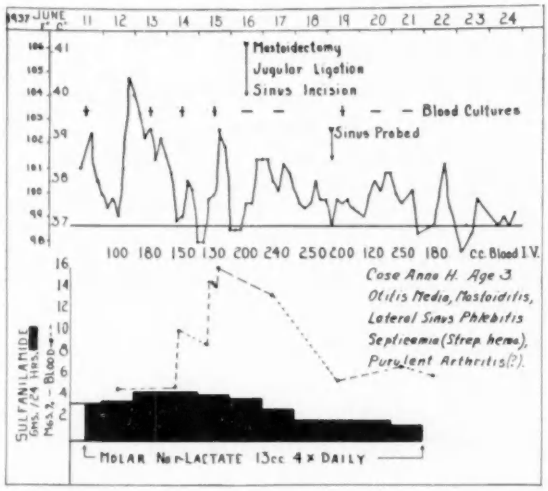


FIG. 2 — (Hartmann)

Figure 2 shows the first of those two.

There are two things to which I want to call your attention. Sulphanilamide was started on the first day of admittance to the hospital because the picture was that of sepsis. The blood culture was positive. Sulphanilamide was given in dosages larger than usually advocated. The dosage is usually a tenth of a gram per kilo per day. Here we have given three-tenths of a gram per kilo — three times the usual recommended dosage. We continue at about the same level with sulphanilamide administration, follow the concentrations of sulphanilamide in the blood, and reach a peak of 16 milligrams per. Daily blood cultures were taken, and you will notice they were still positive four days later, but negative at the

time of operation and continued to be negative except for one transiently positive culture after the sinus was probed. Also notice that every day the blood transfusions were given.

We have a lot of other data on this case which would be nice to put in here, but would clutter up the chart so that you would miss the essential points. The amount of blood given during the course of about two weeks was about twice the normal blood volume, and that much blood seemed to be necessary to prevent the development of anemia from the combined effects of the drug and the infection and particularly to keep a satisfactory concentration of oxyhemoglobin present. We determined quantitatively the amount of total hemoglobin pigment as well as quantitatively the amount of oxyhemoglobin present, and did not permit, by the aid of these transfusions, hemoglobin capable of carrying oxygen to drop to a point below 10 grams, which we think is the critical line.

PRESIDENT DEAN: May I ask a question? At what point was the diagnosis of septic thrombophlebitis of the sigmoid sinus made in this case?

DR. HARTMANN: In this case it was made on the second day. The blood culture was positive. The Queckenstedt test here was positive. The mastoiditis was proved without any question, and the whole picture was thought very typical of thrombophlebitis.

PRESIDENT DEAN: I should like to raise with this group the question of postponing the operation for one, two, three — how many days?

DR. HARTMANN: Three full days.

PRESIDENT DEAN: Three full days, while the hemotherapy was being given. Does that meet with the approval of this group?

DR. KOPETZKY: It would not meet with the approval of my outfit.

PRESIDENT DEAN: Then I think this is something that should be discussed.

DR. TOBEY: Was this postponed on the advice of the pediatrician or on account of the condition of the child?

DR. HARTMANN: With full agreement of the otologist.

PRESIDENT DEAN: May I say that in our group Dr. Hartmann is chairman of the Department of Pediatrics, and that we treat no case of otitic sepsis without the aid of the pediatrician. We have found that it pays to be guided by their advice in the timing of operations which, at least in the past, we have thought were emergency ones. My impression is that I had nothing to do with this case. Nevertheless, as Dr. Hartmann has stated, the operation was postponed after consultation either with Dr. Hartmann or some representative of the Department of Pediatrics.

I think it is very, very important to discuss whether or not we should wait and note the effect of the administration of sulphanilamide or whether we should proceed immediately to the operation. What is your reaction, Dr. Tobey?

DR. TOBEY: I should operate.

DR. LILLIE: So much has been claimed for sulphanilamide, I think this is a good example of why we should not depend alone on hemotherapy in this type of disease. I am in sympathy with Dr. Hartmann as regards consulting otologists in this situation. If this child came in on this first day, and even though a diagnosis of septic thrombosis of the sigmoid sinus was made, the pediatrician might want to know considerably more about the general condition so that he could help along in the postoperative management of the case.

Then there is another thing that I notice in the chart that may have been an influence. There were four positive blood cultures. After the administration of sulphanilamide, in fours days blood cultures were negative. I am glad that did not prevent him from operating upon this mastoid condition. I would not like to be arbitrary and say that I would have operated the second day.

PRESIDENT DEAN: May we discontinue the discussion until we have some other cases before us, because we may want to come back to it again?

DR. HARTMANN: Now this is our only other case of lateral sinus phlebitis treated with sulphanilamide, — younger, only two years of age; pretty much the same findings are to be noted (Fig. 3). Sulphanilamide was started with one large dose; then repeated every four hours in smaller amounts. Queckenstedt's test was already suggestively positive here and completely so a few days later;

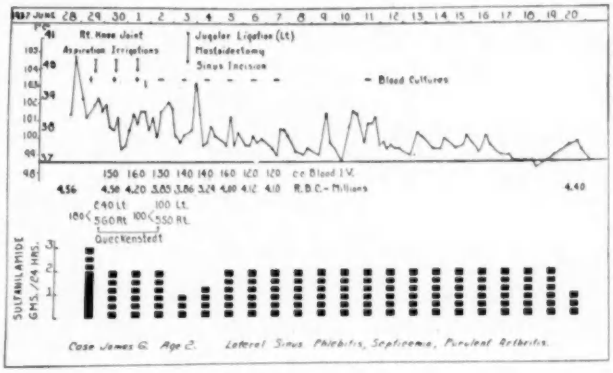


FIG. 3 — (Hartmann)

already a purulent arthritis. I think this is an interesting thing. Before operation, before the blood cultures became sterile, which also became sterile before operation, the knee was aspirated and pus containing the hemolytic streptococcus was secured. Two irrigations with sulphanilamide were resorted to, and the joint was then sterile, before operation. Whether that was local effect or partly local and partly the result of high blood concentration of sulphanilamide that had been built up at this time, of course, one can't say, but here again are two negative blood cultures before operation, and here are the postoperative negative blood cultures. We continued with daily administrations of citrated whole blood, about 10 c.c. per kilo. The erythrocytes did drop to 3¼ million right here, but for the most part kept up to 4 million or above.

DR. LILLIE: Was this a sinus phlebitis or a thrombosis?

DR. HARTMANN: Diagnosis in the first place was very hard to make. Both ear drums showed practically nothing. X-rays in this case were not much help at all, and a very firm clot occupying a good part of the sinus was found already to have broken down—

DR. KOPETZKY: I should like to ask, were the knee joint symptoms present when the case came under observation?

DR. HARTMANN: Oh, yes.

DR. KOPETZKY: Were the knee joint symptoms present in the first case, too?

DR. HARTMANN: In the previous case you noticed a question mark about the presence of arthritis. We never were sure because we did not tap the joint. There was pain in both hips and limitation of motion. Sometimes it is very hard to say just what is in a joint or around a joint in a case like this.

DR. KOPETZKY: A very good deduction can be made from both these cases. This chemical, sulphanilamide, if you use it with the focus intact, you clear the blood but you do not control the antecedent lesion. These cases came in with thrombosis or phlebitis and secondary lesions of the joints, and irrespective of anything else, it would not have taken much time to eradicate the whole primary lesion fully, and then make your studies and perhaps save the necessity of opening into the knee joint even for puncture.

PRESIDENT DEAN: Dr. Hartmann, am I right in saying that there you have had a very severe case of pneumococcus meningitis with an infected ear and a case of hemolytic streptococcus meningitis that recovered. Will you briefly mention those two cases?

DR. HARTMANN: In the first place, I should like to say just a little more about the way we feel about sulphanilamide at the present time. I would not any more think of permitting a child like this to get out of the hospital without a mastoidectomy and a sinus incision that I would do anything. The really red hot infections are made to quiet down, and you can see that so beautifully in erysipelas, whereas if you use the drug the temperature falls by crisis and the redness of the skin disappears, but if you stop the drug too soon, a sort of cold abscess develops from which you can still culture organisms.

We do not know how long we would have to give such a drug to eradicate this infection completely without good adequate surgical drainage, and not knowing that, not knowing it is possible even, would make us want to operate at the proper time; but I believe in this condition, as in many others, one can operate too early, before the patient is in as good a shape as you can get him. There is no reason to delay it unnecessarily, but one cannot do things to babies or children in just six or eight hours when changes of a metabolic nature have occurred which require two or three days for correction. In addition to a recovered case of hemolytic streptococcus meningitis we have had two other cases of meningitis which were extremely interesting. One was a type 5 pneumo-

coccus, disseminated, perhaps of otitic origin, — at least there was a history of mastoiditis involvement preceding the meningitis. The other one turned out to be due to the bacillus proteus. Both of these children were admitted with disseminated meningitis and in dreadful shape. Both of them were treated with sulphanilamide parenterally and were freed of septicemia and meningitis, and only later were their mastoids operated.

PRESIDENT DEAN: Have you another case along the same line that you want to show before we continue this discussion? I think that we have tried and formed either a unanimous opinion as to just how this drug should be used, how long it should be used before operation, or have stated quite divergent views regarding the matter. It is easily conceivable that we can have divergent views with a new drug, as this is. Nevertheless, I think we owe it to ourselves, and you owe it to me and to the rest of the people in the audience, to give an exact conception of any divergent views that may exist.

DR. KOPETZKY: We have used this drug extensively on all our sepsis cases this winter. I did not think we were going to have a discussion of medical or chemical treatment of meningitis, but I will say in passing that we have been successful in meningeal cases this winter with the use of that drug after thorough surgery and assisted by repeated small blood transfusions. The Board of Health in New York City has had a marvelous experience with hundreds of meningeal cases with a better mortality rate than they have ever been able to attain heretofore in recoveries.

DR. HARTMANN: You are speaking about meningococcus meningitis?

DR. KOPETZKY: Meningococcus, streptococcus and pneumococcus. In all our lesions, my routine is as follows: As soon as we make a diagnosis, and locate a purulent lesion, that lesion is attacked surgically and removed; concomitant with the work-up of the case, and during the postoperative period until that case is normal, if it lives, the administration of the drug as an additional measure to all of our hitherto accepted procedures are carried out. These consist of transfusions and other sustaining measures. We have one thing that we must watch in some of our meningitis cases. In the late stages we got concentration of hemoglobin, and we di-

lute the blood plasma with small transfusions of plasma alone, sometimes because we had a hemoglobin index report of over 100, 110 and 115. Those were in cases that recovered.

PRESIDENT DEAN: I think we should avoid discussing the deleterious effects of sulphanilamide at this time, as that would probably take two hours or more. Dr. Kopetzky, I brought in the discussion of hemolytic streptococcus meningitis because it seems to me that would have some bearing on the statement that I understood you to make that its beneficial action resulted because you are dealing with a local lesion. So I brought into the picture lesions which seemed to me to be quite diffuse.

DR. KOPETZKY: We have used both methods. We have not discarded adequate surgery, and have removed the focus, going to the extent of proceeding with the entire eradication of temporal bone in otitic meningitis cases, and they have recovered.

PRESIDENT DEAN: May we now briefly discuss one question: Is it advisable to postpone the operation on the sinus, as it was postponed in these cases, one for four days and the other, how many days, Dr. Hartmann?

DR. HARTMANN: On both, five days, but the diagnosis wasn't made with certainty until about the third day.

PRESIDENT DEAN: Or should we operate immediately or within twenty-four hours, or very soon after the diagnosis is made?

DR. KOPETZKY: May I ask you a question, sir? Obviously, we are looking at this case backwards, ex post facto, — but even from that point of view, with a developing lesion and a gravely sick patient, do you believe it is better to allow the lesion to progress, metastases to be developed, while you are putting the patient in condition, when you are only missing probably a half hour of that patient's life when you proceed immediately to eradicate a major lesion in the sinus and then go on with your other treatment and condition your patient?

DR. HARTMANN: In these two instances the children were getting better during the time the operation was postponed. The knee joint, for instance, in one, became sterile, the blood cultures were becoming sterile, and no further metastatic foci were occur-

ring. Shouldn't you consider also the stage of the mastoid infection? Suppose you see a child after only one or two days of mastoid involvement, while he still has a very acute tonsillitis and otitis and bronchitis, and suppose you have every reason to believe that he is beginning to develop phlebitis, although I do not know how you could be so sure about it at the time, would you want immediately to clean out that mastoid? Wouldn't you want to try sulphanilamide first?

DR. KOPETZKY: I do not want to be understood as criticizing the management of this case, and I do not want to be understood as laying down a rule for all such cases. In answer to your question, I believe that when the diagnosis *is made* and you are positive, reasonably sure, that you have a septic thrombus or a septic phlebitis, or thrombophlebitis, I think the sooner that is eradicated the better. I doubt if in working on this case, with attempts to make the blood sterile and yet the metastases occurring in another knee or in the shoulder right while the case was under observation would make one feel very comfortable. All that time is lost in attempting medication while the lesion that you know is there in the sinus. There is too little that the pediatrician can do in putting the patient in the finest condition, and bringing him back to health; and it were very much better to let the surgeon get rid of the major lesion, and then let the pediatrician take charge and recondition the patient.

PRESIDENT DEAN: For two years before we knew of sulphanilamide, we had been postponing our operations upon what we considered to be diagnosed cases of septic phlebitis of the sigmoid sinus. Whether this has had anything to do with the decrease in the mortality or not, it would be a little hard to say, considering the number of cases at our disposal. It is true that since we were less hurried in going into the mastoid, exposing the sinus and operating, before we had the use of this drug, we felt that we were getting better results. I do not know whether or not that is Dr. Hartmann's impression. At any rate, each case must be approached as an individual case, with the pediatrician on one side of the bed and the otologist on the other side of the bed, and after a serious consideration of the case, I think that I may state frankly that we have been much less in a hurry to go into the sinus. At any rate, we have not gone in until there has been agreement on the part of

the two partners concerned that the sinus should be entered. We are particularly prone to delay if the patient is improving.

Dr. Furstenberg, we want to have a little experience meeting here. What is your reaction in this matter?

DR. FURSTENBERG: I am obliged to lay down the following rule as a surgical axiom. When a diagnosis of septic sinus thrombosis is made, we are justified in using every therapeutic measure at our disposal, and the two now under consideration are, first, an operation, and second, the use of sulphanilamide. I would be inclined to agree with Dr. Kopetzky. When the diagnosis is established, use both methods of treatment.

DR. TOBEY: I have to agree with Dr. Kopetzky and Dr. Furstenberg. We may have to change our conception of the treatment of these cases when we have had more experience with the chemical therapy, but at the present time I should certainly feel that the surgical principle should still be followed.

DR. LILLIE: Now we are talking about generalities. Here is a patient who is very sick and washed out, if you immediately operate on that patient, you are doing more harm than if you waited twenty-four or forty-eight hours, in order to improve the general condition by using fluids and blood transfusions. Give these patients a good rest. They sometimes come from great distances and are fatigued. I know that has been my experience and since that regime has been followed I have not regretted it. If the patient is not too ill and you are not firmly convinced that your diagnosis is right, you should wait, because there are other things that may develop in his illness that will clear up the diagnosis. A central pneumonia and the pyelitis are the most common conditions. Physical examination of the chest may be misleading.

We started with the premise that this patient had acute respiratory infection with subsiding tonsillitis with a possibility of pneumonia, and a general septicemia manifested by focal infection in a joint and ear. It must be determined which is the causal factor.

DR. TOBEY: Aren't you getting away from the original question? As I understood the original question, having a positive diagnosis of lateral sinus thrombosis, septic thrombosis, with or without metastases, and now you are getting away from the case with a problematical diagnosis —

DR. KOPETZKY: We are not in disagreement.

DR. HARTMANN: No one disagrees with that at all.

DR. LILLIE: If I am on the spot in that respect, I will say that if I know I have a diagnosis of infected thrombus in a sigmoid sinus and my patient is not in good physical condition, I will delay twelve to twenty-four or thirty-six hours, to give him fluids and to get him in shape, because I feel from my experience that he will withstand the operation better and will do better following.

PRESIDENT DEAN: I will try to state our position in the matter briefly, namely, that every child who comes in is placed in the hands of the pediatrician to get him ready for the operation, and we do not hurry about it. We go home and get a good night's sleep. If it is evident, perhaps at the end of twenty-four hours, that this patient is on the upgrade instead of on the downgrade, we procrastinate.

Briefly stated, we are influenced by what happens as the result of the pediatric treatment. If these children continue to improve, whether it is meningitis or thrombophlebitis of the sigmoid sinus, we move very slowly as long as the child is on the upgrade. To sum the thing up, we are not in a hurry with any of these cases, as we were a few years ago, and, as I said, our mortality has improved since we have been proceeding on this basis, but the improvement might be due to other factors.

DR. LILLIE: Adults also have this disease, and I was talking about both adults and babies.

PRESIDENT DEAN: Well, I think that everyone in the room knows the opinion of the various men about the table. Now I will be perfectly frank in saying that the panel discussion is my baby, and it was born and reared because I felt that there was bound to be divergent views, that we are entitled to know what they are at this time, and that they would be of some assistance to you when you get a patient with this disease.

Have you anything further on the medical treatment?

DR. HARTMANN: I don't think so.

PRESIDENT DEAN: If not, we will hear from Dr. Brown.

Panel Discussion

SEPTIC THROMBOPHLEBITIS OF THE SIGMOID SINUS

Surgical Treatment

JOHN MACKENZIE BROWN, M.D.

LOS ANGELES

THE SURGICAL treatment of thrombophlebitis of the lateral sinus should be governed by the individual case, and depends upon the existing pathology and the general condition of the patient. The procedure of choice varies considerably in the minds of otologists, as revealed by the literature on the subject. The treatments herein suggested are given subject to modification as the individual case warrants.

The outcome of any surgical procedure depends upon the skill and judgment on the part of the operator, and the relationship between the serological immunity of the patient and the virulence of the invading oragnism. These factors must be taken into consideration when dealing with this lesion and when considering surgical intervention.

Any patient suspected of having involvement of the lateral sinus should be studied thoroughly before surgery is attempted. There is only one situation where this condition does not permit adequate study. That is when the patient is seen late in the course of the disease. When this occurs, a shorter examination and study must suffice to determine the necessity for immediate surgical intervention.

By far the majority of cases are not emergencies and allow time for thorough study. This should include the following:

1. A complete history, with a study of the middle ear, mastoid and neck region.
2. A complete physical examination, essential in ruling out other possible foci.

3. Neurological and ophthalmological examination.
4. A study of the hourly temperature graph which may reveal a septic curve in cases where there is an active invasion of the blood stream.
5. Chills, if present, are important evidence.
6. The following laboratory studies:
   a. A complete blood count will give an index to the patient's resistance.
   b. Blood cultures, if positive, are of great value and may indicate the necessity of surgical intervention. However, negative blood cultures are not proof of a sterile blood stream. Cultures are more reliable if taken just before or after a chill.
   c. The Tobey-Ayres test is of great value if complete or nearly complete obstruction of the lateral sinus exists. However, if the test fails to show obstructive changes, definite information of the involvement of the sinus wall is not obtained.
   d. X-ray is of value to determine not only the amount of osseous necrosis but the size and position of the lateral sinus and emissary vein. X-ray may also reveal the size of the jugular foramena which may be used as an index to the caliber of the jugular vein.

SURGICAL INTERVENTION

If, after the above thorough study, thrombophlebitis is diagnosed, surgery is indicated.

Three objectives should be kept in mind:

1. Removal of the existing pathology.
2. Adequate drainage of the infected area.
3. Prevention of the dissemination of the infection.

The infected area overlying the sinus is usually drained adequately by a thorough mastoidectomy and removal of the sinus plate. This alone may be sufficient to prevent further spread of the infection.

If, upon uncovering the lateral sinus, there is encountered a fibrinous exudate or granulations, this may well be a protective reaction and does not necessarily imply that pathology exists in the sinus lumen. On the other hand, this pathology of the sinus wall

could be part of a diseased interior. The differentiation depends upon blood cultures and other symptoms of sepsis. Therefore, if one encounters the above pathology without the characteristic signs of sepsis, there is no indication for disturbing the sinus, while if sepsis be present, accompanied by a positive blood culture, one should investigate the interior of the sinus. If sepsis be present with negative blood culture and without recession of symptoms after mastoidectomy and exposure of the sinus, one should not wait too long for a positive blood culture before investigating the interior of the sinus.

If, following a mastoidectomy, a low-grade temperature persists with few blood changes, keep in mind that a thrombophlebitis may be present and may invade the blood stream at any time.

If a perisinus abscess is discovered with little or no wall changes and without sepsis, do not disturb the sinus. If wall changes with sepsis and positive blood cultures be present, the sinus should be explored. A perisinus abscess with sepsis and no wall changes is strongly indicative of pathology in the interior of the sinus and should be watched a day or two for further continued sepsis before opening the sinus.

If an obliterating thrombosis is present which is unaccompanied by sepsis, this must be looked upon as a protective mechanism and should not be disturbed. We have found three cases, one at operation and two at autopsy, in which evidence of previous thrombosis and subsequent obliteration of the sinus was present.

If there be a thrombosis with abscess formation, one must keep in mind that the anterior and posterior parts of the sinus may be completely walled off by a sterile thrombus with adequate protection to the general circulation. The problem is one of local drainage alone.

Should there be reason to believe that the sinus pathology is not adequately walled off, as evidenced by sepsis, positive blood culture, meningeal or cerebral signs, or metastases, the surgical problem is entirely different. In this instance, it becomes necessary to produce an artificial thrombus or barrier. We are confronted with two distinct phases, the anterior and posterior parts of the sinus. The posterior sinus should be exposed until normal wall is encountered and free bleeding obtained. Anteriorly, should there be reason to believe that disintegration of the thrombus has stopped short of the bulb, ligation of the jugular vein may well be omitted, at least for the time being. Should the process extend into the bulb, then a ligation of the jugular is indicated.

After incising the sinus wall and removing the thrombus, one must decide whether a simple packing will suffice or whether a portion of the wall should be removed. The wall should be excised if necrotic. If not, a simple packing of gauze or of rubber tissue will suffice. The wound should be left wide open. The packing should be removed gradually and a little at a time. At the initial removal of the packing it is well to have adequate assistance and be prepared for the eventuality of a severe hemorrhage.

A type of sepsis occurring early in a mastoiditis due usually to an extension to the sinus from phlebitis of tributaries in the mastoid calls for early intervention consisting of mastoidectomy and uncovering of the sinus. If definite wall changes are found, the sinus should be opened. If normal wall is found, one may wait for the result of mastoidectomy, blood transfusions and other therapy. Should sepsis continue, with or without positive blood cultures, opening the sinus is in order, despite the normal appearance of the wall. The development of metastases justifies the immediate investigation of the sinus. If upon incision of the sinus wall a mural thrombus is encountered which does not extend into the bulbar region, all that is necessary is the free incision and packing of the interior of the sinus and the blocking off of the two ends.

Should the septic symptoms persist despite the adequate drainage of the sigmoid sinus, then we must suspect an extension of the process. This could be in the direction of the bulb, the torcular, the petrosal or the emissary veins. The next step is the ligation of the jugular vein to arrest the dissemination of any infection in the bulbar region and investigation of the sinus toward the bulb and the torcular, including the superior petrosal and the emissary veins. Several cases are reported in which septic thrombi have been removed from the superior petrosal and the emissary veins. We have had four cases in which it was necessary to proceed as far as the torcular and even to the superior longitudinal sinus. Also the possibility of a double lateral sinus should be kept in mind as in a case reported by the writer.

THE JUGULAR BULB AND ITS TREATMENT

Primary jugular bulb infections are not common but occur in a certain percentage of cases, especially in children and in chronic otitis media. They should be treated in the same manner as bulbar extensions of phlebitis of the lateral sinus. The majority of phlebitic involvements begin in the sigmoid area and the minority in the

bulbar region. If the infection has invaded the region of the bulb, the sinus wall should be uncovered well forward until free bleeding is obtained. The sinus is packed off with gauze, keeping that portion of the sinus wall back of this open with gauze or rubber tissue. One may remove a portion of the wall. If the wall is markedly infiltrated or soft, we suggest the latter procedure. If the thrombus extends into the bulbar region, as much as possible should be removed with a curette or by gentle suction. Then a plain gauze trailer should be inserted as far as possible toward the bulb.

The next step is to expose the jugular vein in the neck. The procedure to follow again depends upon the local pathology found. If the vein is thrombosed or has a diseased wall, one should go below the thrombus and ligate distal to the diseased portion, bringing the upper end of the vein to the neck wound.

If the vein is found to be normal it should be tied with two ligatures above the facial and cut between, and the upper end brought out to the wound in the neck. If the bulbar region is not adequately drained through the sinus, it may then be drained by this upper segment, and it may serve as a channel to irrigate the bulb. These proceedings seem to be much more logical than a direct attack upon the bulb. In cases of complete thrombosis of the bulb area, where one is unable to obtain free bleeding or drainage from the bulb and where sepsis continues and all other areas of infection have been eliminated, one is justified in attacking the bulb.

BILATERAL MASTOIDITIS AND THROMBOPHLEBITIS

The following may be helpful in arriving at a diagnosis:

1. The history — the first ear to be infected is the more probable one.
2. There may be more extensive bone destruction on the affected side.
3. The appearance of the wall may suggest one side more than the other.
4. The involved sinus usually casts a sharper shadow than is cast by the uninvolved side by x-ray.
5. The jugular region on the affected side may present some symptoms in the neck.
6. The Tobey-Ayres test.
7. The Crowe-Beck test.

8. Eye examination — a papilloedema may be present in the affected side.
9. Paralysis of the abducens nerve on the affected side.
10. If sepsis continues and one side has been opened, the other side may be opened after several days have elapsed to allow for readjustment of the circulation.
11. If both sides appear normal and the foregoing tests have not made differentiation possible, the situation is a very difficult one. Here, medical treatment should be carried out to the limit.

THE SURGICAL MANAGEMENT OF THROMBOPHLEBITIS SECONDARY TO THROAT INFECTION

In the presence of sepsis and a throat infection, drainage of the infected area is indicated and especially the investigation of the submaxillary, parotid and pharyngo-maxillary spaces.

If there be positive blood cultures, repeated chills or metastases, the carotid sheath should be exposed for some distance and opened for inspection.

If the jugular is found thrombosed, ligate below the pathology and remove the vein to beyond the thrombus above, tying all tributaries. If the thrombus extends to the bulb, ligate as high as possible, and if the sepsis continues, the mastoid and lateral sinus should be investigated and treated as hereinbefore stated.

If the vein shows a diseased wall and no thrombus, ligate below the pathology and wait for continued sepsis before resecting the vein.

If you find normal appearing vein with sepsis and have drained early and the patient is in good condition, wait for the result of drainage. If no improvement in a few days, ligate the jugular. If there be positive blood cultures, repeated chills or metastases, ligate the vein at once.

CONCLUSIONS

1. Sepsis in the presence of a mastoiditis or throat infection should mean careful and continued study of the patient.
2. Other possible sources of the sepsis should be excluded.
3. The general symptoms are the indications for surgery, while the local findings are the guides for the operative procedure.
4. Should there occur in the course of an otitis or throat infection, sepsis with repeated chills, positive blood cultures or

metastases, there is a positive indication for investigation of the lateral sinus or jugular vein.

5. In the thrombophlebitis due to continuity of diseased tissue and marked by local pathology, drainage of the infected area may be all that is necessary to arrest the process.
6. The other type due to an extension from the tributaries of the sinus or vein and exhibiting few or no local signs of pathology, demands an early blocking of the sinus or vein.
7. Lastly, but by no means the least, the ligation of the jugular vein has been so consistently a factor in arresting sepsis after other procedures have failed that we firmly believe that when indicated, morbidity and mortality is markedly lessened thereby.

* * *

PRESIDENT DEAN: Dr. Furstenberg, will you make such comments as you think best on Dr. Brown's presentation? I should particularly like to get an expression as to just what should be done when it is necessary to ligate the jugular vein; just what you do.

DR. FURSTENBERG: I realize that there are many different opinions concerning the ligation of the jugular vein. I quite agree with Dr. Brown that when the thrombus extends down into the bulb, doubtless it has gone beyond any limit that can be reached from above with the curette or with gentle suction. It therefore becomes necessary to ligate the jugular vein, and when this is done nothing is lost by bringing the upper end of the vein to the margins of the wound. It is sutured to the skin and 3 or 4 days later an attempt is made to irrigate the vein through and through from above.

It seems like good surgery to me to make some effort at least to drain the infected thrombus which remains within that segment, composed of the distal end of the sigmoid sinus, the cephalic end of the jugular vein and the jugular bulb, and sometimes this can be accomplished successfully by through and through irrigation. I think one often sees a rapid drop of temperature and general improvement in the patient following the successful irrigation of that segment which continues to harbor infected material.

DR. TOBEY: I am still old-fashioned enough to tie my jugulars, and I feel very strongly on the subject. In the old days we used to

see these cases with extension of thrombi down into the jugular and below which required very extensive operating, and in about 1923 or 1924, when ligation came in, we discontinued to see the cases. I am not at all certain that anyone is able to say whether or not there is a phlebitis extending downward along the jugular vein, and tying early, we do know that a simple ligation will stop the progress of an extending phlebitis. Personally, I am not so keen on cutting the vein, I mean the vast majority of them. If you simply ligate and replace it and close your wound, the vast majority will get well without a secondary infection in the neck. Of course, if there is evidence of a breaking down thrombus in or below the jugular bulb, the procedure of bringing the proximal end to the surface is very easily carried out.

I was rather surprised at the statement of the reader that primary thrombosis of the jugular bulb is more common in chronic suppurative ears. My experience has been that they are much more common in children, occurring, of course, in adults; and many times during the course of a very mild infection of the middle ear, without a surgical mastoid. In my own family, or on myself, I still feel that having a diagnosis of lateral sinus infection, thrombosed or not, I should insist on tying the jugular vein, and I know that I should feel very much more comfortable.

Dr. Lillie: I think most of you know my position. It depends entirely upon the stage of the disease and the type of pathologic lesion. In the old days, in 1904, of which I have no recollection from the standpoint of surgery, it is true that jugular vein ligation and section and dissection were used because those patients were seen in the end stages of the disease. It is reasonable to think that if one can get in ahead of that stage, less surgical interference needs to be done. I know it is borne out in my own experience. If I have a patient in extremis — for instance, I can briefly recite a case of a man about forty years old who was sent to us. He came in and, peeling off three or four suits of clothes to protect himself from the winter, he fainted. His partner who was with him said he had been treated for typhoid fever for six weeks. An examination revealed a chronic suppurative otitis media and mastoiditis. Immediate operation revealed suppurative labyrinthitis, cerebral abscess, sinus thrombosis, and a thrombus extending down into the jugular vein. Such a case demands adequate operation. I am in accord with the position that Dr. Brown has stated, that what the

surgical interference should be depends upon the stage of the disease, the condition of the patient, and the pathologic process encountered and not the reverse.

DR. KOPETZKY: Dr. Dean, I find myself happily in accord with what everybody has said. I think that if we differentiate the cases, and have no preconceived rules, but individualize, we will certainly handle better the cases that we receive late and that are in extremis. They need much more vigorous treatment than those seen early.

I should like to close by making some comments on Dr. Brown's treatment of retrograde thrombosis from streptococcus infected tonsils. I am in accord with the outline of the treatment that he made there with this addition, that you are only establishing drainage at the beginning of the retrograde end. You have not opened the proximal end where the infection starts, and, following the European custom, I have had best success with them by opening that end, namely, I take out that infected tonsil, in the few such cases I have had, then I put in a mattress suture to control the hemorrhage, then rapidly curette the area, and then tie the sutures so that now I have both ends of that retrograde thrombus confined within sutures. After that, give all the supporting treatment that you can.

There is a very rich literature growing up on these cases of retrograde tonsillar thromboses, and when the lesion reaches the jugular, if it gets that far, jugular ligation is absolutely necessary, and in the study of the bilateral ligations of those jugulars that I have seen, these tremendous heads and distortions that I talked about were observed, it is among such cases that I got the information that I spoke about in the earlier part of this symposium.

PRESIDENT DEAN: Dr. Hartmann, how would you operate?

DR. HARTMANN: That is a bad question, but it seems to me that no matter how you operated, you could not excise or you could not stop or remove or eradicate, or whatever you wanted to say, all the infection. There is going to be something left, with the possibility of extension of infection. It does seem to me — and I have been guided largely by Dr. Lyman's ideas and Dr. Dean's ideas more lately — that, especially in the stage of the mural thrombus, when there is circulation of the blood still going through the vein, you can take off a big load from the body by ligating that jugular

vein. You can't take it all off, but you can take a lot off, and maybe just enough to let your patient live.

I agree with Dr. Tobey, that if I had a lateral sinus infection, thrombosed or not, I should like to have the jugular ligated to give me as much chance as possible.

DR. BROWN: In reply to Dr. Tobey, regarding primary thrombosis of the jugular bulb, I did not mean exactly what he understood. Most of them are in children, and most of them in adults are from trauma.

I should also like to ask Dr. Kopetzky, would not the drainage of the pharyngeal maxillary space do much good by removal of the tonsil?

DR. KOPETZKY: The other is easier, and attacks the lesion at its source.

PRESIDENT DEAN: I wish to thank the gentlemen about the table for helping me with this panel discussion. The time is up. The business meeting will be on in a few minutes, and we will adjourn.

Instructional Subjects

The American Academy
of Ophthalmology and Otolaryngology
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Forty-Second Annual Meeting
Chicago 1937

Instructional Subjects

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* * *

C. ANATOMY OF THE EYE AND ORBIT

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BY INVITATION

* * *

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J. A. BABBITT, M.D., Philadelphia
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   B. *Ankylosis of the Lower Jaw — Anthroplasty*

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    c. Pathology of Temporal Bone (colored)

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AN ENUCLEATION COMPRESSOR*

D. H. ANTHONY, M.D.

MEMPHIS, TENN.

THE instrument described in this paper is used during enucleation. Either the large or the small end is inserted into Tenon's capsule immediately after the optic nerve has been severed. By applying pressure in the apex of the orbit one can compress the bleeding vessels of the orbital apex. This pressure will immediately stop the bleeding.

This instrument is held tightly against the apex of the orbit while the sutures are inserted in Tenon's capsule. The handle is made 10 inches (25.4 cm.) long in order that the assistant's hand

The enucleation orbital compressor.

holding the instrument may be out of the way of the operator. The shaft of the handle is small (6 mm. in diameter), so that it does not interfere while the operator is inserting sutures in Tenon's capsule.

The tips of this instrument are olive shaped; the large end measures 14 mm. and the small 11 mm. The large end is useful for operations on adults and children, and the small end is useful for operations on children under the age of 5 years.

* Made by V. Mueller & Co., Chicago, Illinois.

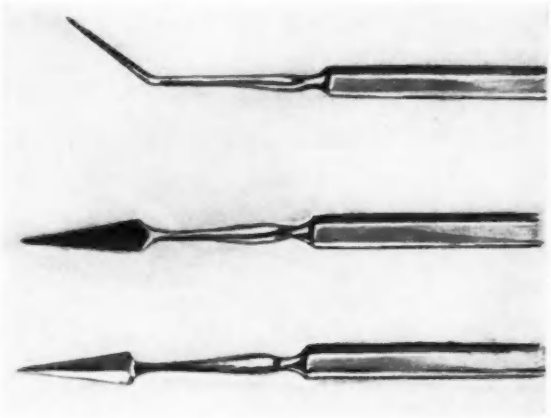
A NEW KERATOME*

A Hollow-Ground Narrow Angular Keratome for Paracentesis, Iridocapsulotomy and Iridotomy

CONRAD BERENS, M.D.

NEW YORK

THIS keratome was constructed because of the desire to have a long narrow keratome which would penetrate the eyeball and secondary membranes with as little trauma as possible.



Description. — The handle is 95 mm. in length by 4.5 mm. in diameter. The shaft is 27 mm. in length and the blade is 19 mm. in length, and 3.25 mm. broad at the base. The angle of the blade and the shaft is 140°. The undersurface is hollow ground and the upper surface is slightly ridged to add strength.

Advantages. — (1) The hollow grinding permits the development of a razor-keen edge.

(2) The incision made by the hollow-ground blade tends to close rapidly, preventing loss of intraocular fluids after iridocapsulotomy.

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* Made by V. Mueller & Co., Chicago, Illinois.

(3) The long narrow blade permits incision of the iris or capsule at any desired distance from the primary incision without making the corneal or scleral incision too large.

(4) The exceedingly sharp point and blade permit penetration of the iris and capsule without too great traction.

A NEW TYPE OF MADDOX ROD*

CONRAD BERENS, M.D.

NEW YORK

THE ROD was developed because of the desire to obtain an inexpensive, indestructible rod for clinic and office use. The material used, although it has practically the same index of refraction as glass, cannot be broken or chipped. The rod is 40 mm. in length, 22 mm. in horizontal diameter and has a radius of curvature of 13 mm.

The rod is made both with and without a prism. The apparatus with the prism has a ¾△ with the base in line with the long axis.

The Use of the Rod. — When the long axis of the rod is placed vertically before the eye, a horizontal streak of light is seen as with the Maddox rod. When the vertical diameter of the rod is held transversely, a vertical line is seen, and when the rod containing the prism is used, by rotating the rod through 180°, low degrees of hyperphoria, esophoria, and exophoria may be rapidly determined. For example, if there is no hyperphoria and the rod is

* Constructed by E. B. Meyrowitz Company, 520 Fifth Avenue, New York.

placed vertically before the right eye with the base down, the streak of light will be seen first above the light and then below the light, and the patient will state that the distance of the light streak from the light is the same when the rod is held in these two positions. If there is $\frac{3}{4}^{\Delta}$ of right hyperphoria, the patient will say that the streak image is through the light when the prism is base down before the right eye and the streak image will be displaced markedly below the light when the rod is rotated through 180°. To facilitate the examination in a lighted room or a busy clinic, the light source may be red, which will differentiate the streak made by the rod from those made by other lights.

CAPSULE FORCEPS FOR INTRACAPSULAR CATARACT EXTRACTION*

FREDERICK ALLISON DAVIS, M.D.

MADISON, WIS.

THESE FORCEPS have a smooth biting surface and are so designed that a very large area of the anterior capsule of the lens can be grasped with a minimum danger of tearing. The blades are long, broad, and smooth, so that a very large surface is in contact with

Capsule forceps for intracapsular cataract extraction.

the fragile capsule. The jaws of the forceps, just back of the approximating surfaces, leave a gap or space through which the capsule can protrude. The approximating surfaces are so long that a large area can be grasped, thereby distributing the pull or traction throughout the entire circumference of the lens, thus lessening the danger of tearing. When a small, pointed instrument is applied to a more limited area, danger of tearing is increased. The angle of the forceps permits an easier entrance to the anterior chamber than is possible with some of the longer and more oblique models. The lens may be delivered by tumbling or in the upright position, depending upon the nature of the cataract. The instrument is suitable for use with any of the methods employing the forceps expression technic.

* Made by V. Mueller & Co., Chicago, Illinois.

A METHOD FOR TESTING THE VISUAL ACUITY OF CHILDREN

WALTER H. FINK, M.D.

MINNEAPOLIS

THE METHOD is a modification of the "E" test which is commonly used for testing the visual acuity of children. It differs in that the letters are placed upon a disc of white drawing paper, 8 inches in diameter, and has the advantage that it can be exposed in any desired position by rotation of the disc.

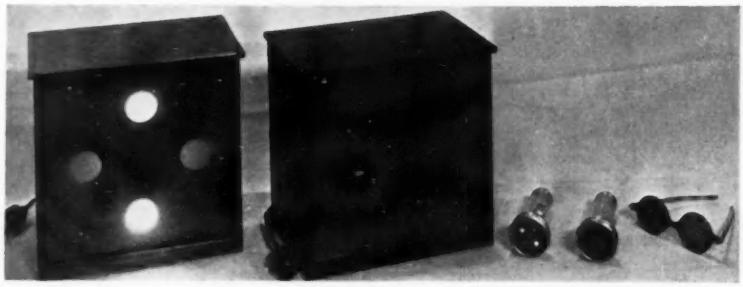
Four discs are used, and on both sides of each disc is placed an "E" of different size, and corresponding to the standard Snellen chart. The vision from 20/200 to 20/15 can be recorded. A plain disc is used as a shield to cover the letter while changing its position.

A MODIFICATION OF THE FOUR DOT TEST

LeGrand H. Hardy, M.D.

NEW YORK

Worth's utilization of Snellen's colored glasses and colored figures to form a fusion test was proved to be a valuable adjunct in his strabismus clinic. We have used this test for several years with much satisfaction.



The modification here presented makes the testing materials easily portable and adaptable for near point work. Five millimeter openings are punched at the corners of a twenty millimeter square inscribed on a circular piece of black paper 50 millimeters in diameter. Green gelatine filters are placed over two holes on one diagonal. A red gelatine filter is placed over one of the remaining holes. The fourth hole is covered with translucent tracing paper. The disc is mounted between a ground glass in front and a clear glass behind and replaces the glass front of an ordinary flash light.

The technical procedures are the same for the Worth Four Dot distance test except that it is no longer necessary to reverse the lenses when checking answers. Several combinations of checks can be immediately and efficiently carried out simply by rotating the flash light.

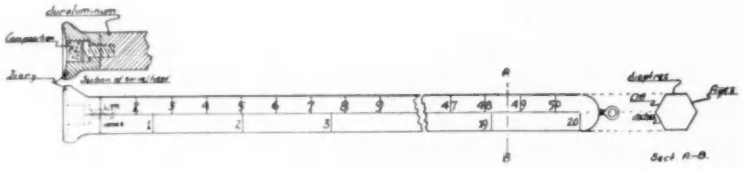
The test is not intended to supplant, or even compete with, Worth's Test as a clinical procedure. It serves merely as an adjunct test at the near point and as a portable test when the larger equipment is not at hand. It is manufactured by Clairmont and Nichols, Guild Opticians, New York.

A MODIFIED ACCOMMODATION RULE

LeGrand H. Hardy, M.D.

NEW YORK

Description: The rule is a modification of that devised by Crampton, the improvements being a swivel head and three new scales.



A 52 cm. hexagonal aluminum rod 1 cm. on edge is fitted on its proximal end with an ivory button 1 cm. in length. This button has a round, expanded base with a concave surface but assumes a hexagonal section at its junction with the rod to which it is attached by a swivel bearing permitting free and independent rotation of either section.

The distal end of the rod is rounded and fitted with a loop or hook for hanging. One face is inscribed in centimeters and the two adjacent faces in ages and dioptres. The age face indicates the average punctum proximum for the age indicated — by twirling the rule this is translated immediately into terms of centimeters, inches (on a fourth face), or dioptres [reciprocals of meters showing the strength of a lens required for the indicated focal length (inches or centimeters)].

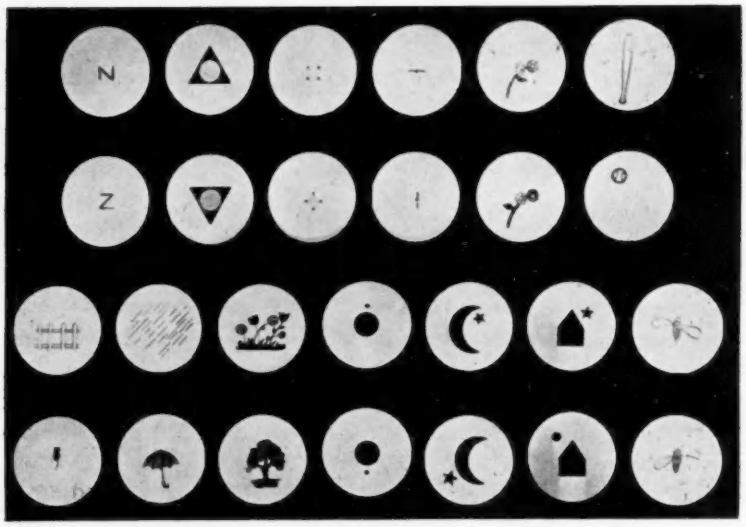
The swivel head permits this manœuvre to be performed without twisting or distorting the skin of the subject's face against which the base (ivory button) rests.

SYNOPTOPHORE SLIDES

LeGrand H. Hardy, M.D.

NEW YORK

I WISH to present to the Academy a series of targets for use in the major amblyoscopes (synoptophore, synoptiscope and orthoptoscope). The reason for presenting them is that effective charts of this type are very difficult to acquire, since their value can be determined only empirically. It is easy to design charts but only prolonged experienced can determine just which ones are effective in serving their purpose.



This series of charts has been selected from a large number designed and used in clinical orthoptics during the past five years for a twofold purpose:

1. To afford accurate measurements of the deviation and of the fusion ability.

2. To facilitate the establishment of normal retinal correspondence — true or normal projection.

Accordingly, they are divided into two groups — a testing group and a training group. The testing group measure lateral, vertical and torsional deviations and frequently gives evidence of aniseikonia. They refine the quantitative measurement of fusion beyond the extent to which it is usually carried. A test for stereopsis is included.

The training group has been selected with great care. It represents the best and most effective of several hundred made and used in clinical orthoptics. The use of vertical and diagonal lines, of silhouettes, and of color stimulation in evoking binocular perception is the result of some research and a great deal of clinical work. The principles used have been proved to be fundamentally correct.

These slides have been designed and selected by Miss Elizabeth K. Stark. They may be obtained through Clairmont and Nichols, Guild Opticians, New York City.

RED-FREE ILLUMINATION FOR BAUSCH AND LOMB BINOCULAR OPHTHALMOSCOPE

H. Rommel Hildreth, M.D.

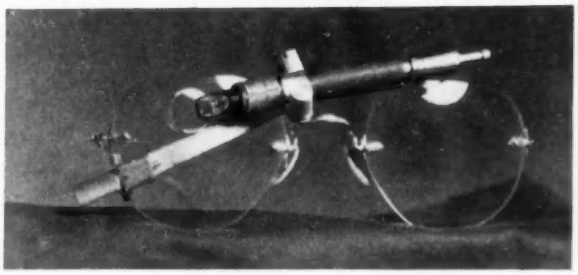
ST. LOUIS

The mercury arc supplies an intense source of light, relatively free from red. Such light as a source of illumination for the ocular fundus accentuates structural details by rendering the vascular tree black against the usual orange background. In addition, the nerve fiber layer becames visible by means of the high proportion of blue and violet rays. A small air-cooled mercury arc operating on alternating current has been adapted to the Bausch and Lomb Binocular Ophthalmoscope.

SURGICAL OPHTHALMOSCOPE

H. Rommel Hildreth, M.D.

St. Louis



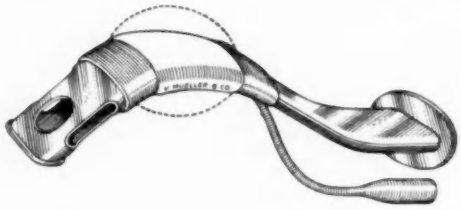
In order to free both hands of the surgeon during a retinal detachment operation and still permit observation of the inside of the eyeball, an ophthalmoscope was reduced to its essentials and mounted on the surgeon's glasses. A simple focussing lens is selected before the operation and its effects varied during the operation by moving closer or farther from the patient. The instrument can be adjusted to either eye.

AIRWAY WITH INFLATABLE CUFF

MARVIN F. JONES, M.D.

NEW YORK

THE OBJECT of this device is to prevent the blood resulting from nasal and oral operations from gaining entrance to the trachea and esophagus. It is designed to replace the use of post nasal tampons or packing of the throat. Trauma to post nasal and aural membranes is eliminated. It may be quickly and easily inflated or deflated without disturbing the breathing tube through which the anesthetic is administered.



The device consists of a rubber collar which, by the power of contraction is held in place around the breathing tube. Superimposed on this collar is an inflatable rubber ring to which a tube is attached. The tube is long enough to project from the mouth and is used to inflate the rubber ring. The inflation is accomplished by connecting a Pomeroy syringe with the tube and pushing the piston to the bottom. A hemostat is then clamped on the tube to hold the air in place. Removing the hemostat immediately liberates the air and deflates the rubber ring.

AN IMPROVED ENDOSCOPIC KNIFE*

MURDOCK EQUEN, M.D., and FRANK NEUFFER, M.D.

ATLANTA, GA.

THESE instruments to be presented are of particular interest to those doing bronchoscopy and esophagoscopy. The necessity of a new or a resharpened blade on each laryngoscopic operation brought to our attention the need of an interchangeable blade just as is used by the general surgeons. With this in view, we designed a small knife with an elongated handle the size and length of which would permit its use through the laryngoscope and esophagoscope. Each time these instruments are used a new blade can be substituted at a small expense.

This knife is of value when the laryngoscope is used in the treatment of infections, which require incision for drainage, involving the larynx or other deep structures of the neck.

* * *

The second instrument to be shown is an esophageal knife. This instrument has been found especially valuable in establishing a lumen through impermeable scar tissue whether the result of the ingestion of a caustic or the prolonged presense of a foreign body. A case in which a child swallowed a bobbin was reported.† An x-ray with lipiodol showed 1 cm. of scar tissue between the esophageal lumen above and the foreign body below. Under biplane fluoroscopic guidance and the use of this knife the scar tissue was cut, the foreign body was pushed into the stomach and was delivered through the gastrostomy opening. This case history is given only to illustrate the value of this knife, however, it is a dangerous instrument in the esophagus and is to be used only under fluoroscopic guidance and with the greatest of care.

* This instrument is made by The George P. Pilling & Son Company, Philadelphia, Pa.

† Equen, Murdock: *Sharp Dissection Through an Esophagoscope,* J.A.M.A. 103:1845-6, (Dec. 15) 1934.

Necrology

The American Academy
of Ophthalmology and Otolaryngology

Forty-Second Annual Meeting
Chicago • 1937

IN MEMORIAM

Charles Franklin Adams, Trenton, New Jersey
George Henry Allen, Topeka, Kansas
Carl M. Anderson, Rochester, Minnesota
Harry Beecher Baker, Taunton, Massachusetts
William Cotterrell Bane, Denver, Colorado
Arthur George Bennett, Buffalo, New York
Marion Earle Brown, New Orleans, Louisiana
Ernest Ustick Buckman, Wilkes-Barre, Pennsylvania
Alphonso Nicholas Codd, Spokane, Washington
Frank Aloysius Conlon, Lawrence, Massachusetts
James Leslie Davis, Philadelphia, Pennsylvania
Ray Arthur Edson, Buffalo, New York
Herschel Charlton Ezell, Nashville, Tennessee
Francisco Maria y Hernandez Fernandez, Miami, Florida
Max Lyon Folk, Chicago, Illinois
Thomas Jones Gallaher, Jr., Denver, Colorado
Frank Yuba Gilbert, Portland, Maine
Samuel Pelman Glover, Altoona, Pennsylvania
Louis David Green, San Francisco, California
Matthias David Haspel, New Orleans, Louisiana
Henry Louis Hilgartner, Austin, Texas
Eugene Eunson Hinman, Albany, New York
Edward Daniel Hurley, Boston, Massachusetts
Arthur Frederick Jackle, New York, New York
Herman Jarecky, New York, New York
John Loring Johnson, Bangor, Maine
Miner Raymond Kendall, Cleveland, Ohio
George Lincoln King, Alliance, Ohio
Samuel Kirkpatrick, Selma, Alabama
James William Leech, Providence, Rhode Island
Lorenzo B. Lockard, Denver, Colorado
Alvin J. Lorie, Kansas City, Missouri
Francis Gabriel Miniter, Boston, Massachusetts
Bernard Anthony O'Hora, Detroit, Michigan
George Oliver Sharrett, Cumberland, Maryland
Elbert Byron Swerdfeger, Denver, Colorado
Milton Thomashefsky, Brooklyn, New York
Charles Christian Walker, Detroit, Michigan
Charles James Wetherbee, Twin Falls, Idaho

Dr. Charles Franklin Adams

Dr. Adams was born July 5, 1859, and died in Trenton, New Jersey, on December 14, 1936.

He received his early education at the Farnum Preparatory School, the Philadelphia College of Pharmacy, and his medical degree from Jefferson Medical College of Philadelphia in 1887.

He went to Berlin for postgraduate study in 1889, to Vienna twice, in 1904 and 1911, and later, in 1914, to Harvard and Rutgers Universities.

Dr. Adams was ophthalmologist to the Orthopedic Hospital, Trenton, and the New Jersey State Home for Boys in Jamesburg, New Jersey, chief of the Department of Ophthalmology and Otolaryngology, and for many years Secretary of the Medical Board of the Mercer Hospital, consultant in ophthalmology to the State Hospital, consultant in ophthalmology and otolaryngology to the Municipal Hospital, Trenton, and the New Jersey State Village for Epileptics, Skillman, New Jersey.

He served during the World War as Chief of the Departments of Ophthalmology and Otolaryngology at Base Hospital, Camp Green, Charlotte, North Carolina.

Dr. Adams was a member of the American Academy of Ophthalmology and Otolaryngology, and Fellow of the American College of Surgeons, and a member of local and Philadelphia medical societies.

He was keenly interested in botany, history and travel.

He is survived by his wife and two adopted sons.

Dr. George Henry Allen

Dr. Allen was born in Harlan, Iowa, on April 18, 1880, and died in Topeka, Kansas, on December 18, 1936.

He received his medical degree from the State University of Iowa College of Medicine, Iowa City, in 1908, and began the practice of medicine in the same year at Clarkson, Nebraska. Later, having taken postgraduate work in his specialties in Vienna and Budapest for two years, he moved to Topeka, Kansas, where he soon became very widely known.

Dr. Allen was on the staffs of the Hillcrest Sanatorium and the Jane C. Stormont Hospital, and was president of the latter association.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member and past president of the Shawnee County Medical Society.

He served in the World War as a lieutenant.

In recent years, Dr. Allen made many extended trips throughout this country and abroad. He was a golf, hunting and fishing enthusiast, and was interested in many organizations, such as Masonic Bodies, and the Elks and Rotary Clubs.

He is survived by his wife, one son and a daughter.

Dr. Carl M. Anderson

Dr. Anderson was born in Waukapa, Wisconsin, on November 7, 1881, and died on August 10, 1937, at Rochester, Minnesota.

Dr. Anderson was graduated from the Medical School of Marquette University, Milwaukee, in 1911; from then until 1921, when he became a Fellow in the Mayo Clinic, he practiced medicine in Wild Rose, Wisconsin.

He was on the permanent staff in the Department of Otolaryngology of the Mayo Clinic from 1924 until his death, and an Assistant Professor in Otolaryngology in the Mayo Foundation of the Graduate School of the University of Minnesota.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Laryngological, Rhinological and Otological Society, and of local and state medical societies. He was also a member of Sigma Xi medical fraternity. His published medical articles were many and valuable. During the World War, Dr. Anderson was commissioned a First Lieutenant in the Medical Corps of the United States Army.

He is survived by his wife, one son and one daughter, a brother and a sister.

Dr. Henry Beecher Baker

Dr. Baker was born in New Bedford, Massachusetts, on June 18, 1858, and died in Taunton, Massachusetts, on June 7, 1937.

He was educated at Boston and New Bedford public schools, the Friends Academy, and received his medical degree from Jefferson Medical College, Philadelphia, in 1880. After an interneship

with the United States Marine Hospital Service, he began the practice of medicine in Taunton, and later specialized in ear, nose and throat work.

Dr. Baker was Councillor and Censor of the Massachusetts Medical Society, delegate to the American Medical Association, consultant to the Morton and Taunton State Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and of various local and state medical societies.

He was a prominent Mason and greatly interested in civic conditions. He was a member of the old Social and Winthrop Clubs and a Rotarian.

Dr. Baker was married in 1883, but his wife died many years ago. He is survived by two daughters.

Dr. William Cotterrell Bane

Dr. Bane was born on November 21, 1855, near Van Buren, Pennsylvania, and died January 20, 1937, at Denver, Colorado.

After graduating from Jefferson Medical College, Philadelphia, in 1879, Dr. Bane began the practice of medicine in Canonsburg, Pennsylvania. Later he took postgraduate work in Philadelphia, New York, and Chicago, and moved to Denver in 1891.

He was associated with the faculty of the old Denver Gross Medical School, the Denver College of Medicine, and the old Denver University Medical School, and at the time of his death was Emeritus Professor of Otolaryngology of the University of Colorado Medical School. He was a member of the staffs of St. Joseph's, Mercy, Presbyterian, and St. Luke's Hospitals; consultant to the Children's Hospital; otolaryngologist to the Denver Orphans' Home; consulant oculist for the Rock Island Railroad and the Denver and Rio Grande Western Railroad.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and the American Laryngological, Rhinological and Otological Society; Fellow of the American College of Surgeons, and a member of local and state medical societies.

In connection with his professional and instructional duties, Dr. Bane studied eye photography and became an expert in color photography in its application to record and teaching purposes.

He is survived by his wife, a daughter and a son.

Dr. Arthur George Bennett

Dr. Bennett was born in Birmingham, England, on September 16, 1861, and died in Buffalo, New York, on December 28, 1936.

He came to America in 1886, and became a naturalized citizen in 1891, in which year he also received his medical degree from the University of Buffalo Medical School, and later took a post-graduate course in ophthalmology in England.

At the time of his death, Dr. Bennett was Professor Emeritus of Ophthalmology at the University of Buffalo; consulting ophthalmologist to the Children's, Deaconess, Lafayette General, Buffalo General, and Columbus Hospitals, Buffalo, and the J. N. Adam Memorial Hospital, Perrysburg; and on the staff of the Craig Colony, Sonyea. He was a member of the House of Delegates of the American Medical Association in 1926.

Dr. Bennett was a member of the American Academy of Ophthalmology and Otolaryngology; Fellow of the American College of Surgeons; and of various local and state medical societies, having been President of the Erie County Medical Society and the New York State Medical Society.

He was a Mason, member of the Lodge of Ancient Landmarks, and author of several articles on ophthalmology in various medical journals.

Dr. Bennett was married on October 7, 1894.

Dr. Marion Earle Brown

Dr. Brown died in New Orleans, Louisiana, on August 19, 1937, at the age of fifty.

He graduated from Tulane University of Louisiana Medical School in 1911, and later became Professor of Ophthalmology to the Tulane University Graduate School of Medicine. At the time of his death, Dr. Brown was on the staff of the Eye, Ear, Nose and Throat Hospital in New Orleans. His service included also the Baptist, French and Charity Hospitals, and New Orleans Dispensary for Women and Children.

Dr. Brown made several contributions of ophthalmic value and was keenly interested in military organizations after his service with the United States Marine Corps in France.

He was a member of the American Academy of Ophthalmology and Otolaryngology and other local and state societies. He was President of the Louisiana Sight Saving Council.

His wife survives him.

Dr. Ernest Ustick Buckman

Dr. Buckman was born at Washington's Crossing (then Taylorsville), Bucks County, Pennsylvania, on August 1, 1863, and died in Wilkes-Barre, Pennsylvania, on June 1, 1937.

He graduated from the Millersville (Pennsylvania) State Normal School in 1884, and took his medical degree from the University of Pennsylvania in 1892, following which he took postgraduate work at the New York Polyclinic Hospital. He began practice of medicine in Wilkes-Barre, was ophthalmologist and aurist to the Wilkes-Barre General Hospital until about 1912, and remained on their Board of Directors until his death, being President of the Board of 1920 to 1930.

Dr. Buckman was a member of the American Academy of Ophthalmology and Otolaryngology and the American Otological Society, Fellow of the American College of Surgeons, and a member of his local and state medical societies.

Dr. Buckman was an active force in the medical life of his community, giving freely of his advice and assistance. Those who knew him will long remember his gracious personality and Chesterfieldian courtesy. He was forceful in conference and consultation, and a most valued friend.

He is survived by his widow, two daughters and two sons, both of the latter being physicians.

Dr. Alphonso Nicholas Codd

Dr. Codd was born August 21, 1894, at Colfax, Washington, and died October 31, 1936, at Spokane, Washington.

He received his preliminary education at Gonzago University and St. Louis University, and his medical degree from Jefferson Medical College in Philadelphia, in 1919, later taking postgraduate work at the University of Pennsylvania Graduate School.

Dr. Codd began the practice of his specialties, particularly endoscopy, in Spokane in 1922. He was on the staffs of St. Luke's Hospital and Sacred Heart Hospital, being Vice-President of the Staff of the latter.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the Spokane Academy of Ophthalmology and Otolaryngology, the Pacific Coast Oto-Ophthalmological, and local and state medical societies.

His interests other than medical were in polo and golf, and he had published some articles on his special subjects.

He is survived by his wife and four children.

Dr. Frank Aloysius Conlon

Dr. Conlon was born in Lawrence, Massachusetts, on May 18, 1881, and died in the same town on June 23, 1937.

He received his early education at Phillips Andover and his medical degree from Harvard University Medical School, Boston, in 1904. Following two years' postgraduate work in Vienna, Austria, he began the practice of medicine in Lawrence in 1906. He was on the staff of the Carney Hospital, Boston.

Dr. Conlon was a member of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons.

He is survived by two sisters.

Dr. James Leslie Davis

Dr. Davis was born on September 11, 1872, on a farm in Jessamine County, Kentucky, and died on August 1, 1936, in Philadelphia, Pennsylvania.

Receiving his preliminary education in the schools of Jessamine County, Kentucky, he graduated from Georgetown (Kentucky) College in 1897, and received his medical degree from Jefferson Medical College, Philadelphia, in 1901, being awarded a prize in each of the Departments of Ophthalmology, Laryngology and Otology.

After an interneship at the German Hospital, Dr. Davis began the practice of medicine in Philadelphia, and was Chief of the Nose and Throat Clinic at the Jefferson Hospital under Professor Kyle from 1903 to 1907, Associate in Laryngology to Jefferson Medical College from 1904 to 1907, and Chief of the Ear Clinic at Jefferson Hospital from 1904 to 1907. At other times he was Professor of Laryngology at the University of Pennsylvania Graduate School of Medicine, consulting otolaryngologist to the Pennsylvania Institution for the Deaf, on the staffs of St. Mary's Hospital, the Philadelphia Lying-In Hospital, and St. Agnes' Hospital. At the time of his death, Dr. Davis was consultant to St. Mary's Hospital and the Philadelphia Lying-In Charity Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and various local and state medical societies.

Dr. Davis was of an artistic temperament, enjoyed travel, and made various contributions to the literature on otolaryngology.

He is survived by two sisters and one brother.

Dr. Ray Arthur Edson

Dr. Edson was born in Crittenden, New York, on April 7, 1882, and was burned to death at his home there on November 5, 1936.

He graduated from the University of Buffalo School of Medicine in 1907, later from Cornell University Medical School, and began the practice of medicine in Buffalo. Later he took postgraduate work in Philadelphia, London, Vienna, and at Dr. Henry Smith's Eye Hospital in India.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and of local and state medical societies. He was Assistant Professor of Ophthalmology at the University of Buffalo School of Medicine, and on the staff of the Buffalo General Hospital.

Dr. Edson was interested in aviation and made several long trips, one from Egypt to India and another from Los Angeles to Philadelphia. At home he was devoted to the beautifying of his grounds and gardens.

He was a bachelor, and is survived by an aunt and several cousins.

Dr. Herschel Charlton Ezell

Dr. Ezell was born on July 27, 1888, near Antioch, in Davidson County, Tennessee, and died on November 26, 1936, at Nashville, Tennessee, where he had lived for many years.

He received his early education at Peabody College, and his medical degree from the University of Tennessee Medical School, in 1911, following which he practiced medicine in Nashville. In 1913 and 1914 he did postgraduate work in Vienna, Austria, and returned to practice his specialties.

At one time Dr. Ezell was on the teaching staff of Vanderbilt University, and was on the staff of the Protestant Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and various local and state medical societies.

He is survived by his wife, a son and a daughter.

Dr. Francisco Maria y Hernandez Fernandez

Dr. Fernandez was born June 18, 1886, and died in Miami, Florida, on February 14, 1937.

He graduated from the University of Havana with the degree of B.S. in 1900, later receiving his medical degree from the University of Havana and the College of Physicians and Surgeons of Columbia University in 1908.

At one time Dr. Fernandez was Assistant Professor of Ophthalmology at the University of Havana Faculty of Medicine, Chief of Clinic, Eye Department, Mercedes Hospital, Havana, and was on the staffs of the Insane Hospital of Cuba, Leprosy Hospital of Cuba, and the Centro Gallego Hospital of Cuba.

He was formerly Cuban Secretary of State and Cuban Secretary of Sanitation, and had been decorated by several governments.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Société Française d'Ophtalmologie, Fellow of the American College of Surgeons, Past President of the Pan-American Medical Association, member of the Mexican Ophthalmological Society, and the Sociedad Oftalmologica Hispano-Americana, Spain. He was also a member of the Havana Academy of Sciences.

Dr. Max Lyon Folk

Dr. Folk died in Chicago on June 7, 1937, aged forty-seven.

He graduated in medicine from the Chicago College of Medicine and Surgery in 1917.

At the time of his death Dr. Folk was attending ophthalmologist to the Michael Reese Hospital, and Research and Educational Hospital of the University of Illinois.

He was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

Dr. Folk served during the World War.

Dr. Thomas Jones Gallaher, Jr.

Dr. Gallaher was born in Pittsburgh, Pennsylvania, on March 17, 1867, and died in Denver, Colorado, on February 5, 1937.

He obtained his early education at Pittsburgh grade and high schools, Elder's Ridge Academy, his A.M. degree from Washington and Jefferson College, Washington, Pennsylvania, and his medical degree from the University of Pennsylvania in 1889. He began the practice of medicine in Pittsburgh, and later, having taken postgraduate courses in Vienna, took up the practice of his specialty in Denver.

From 1897 until 1902, Dr. Gallaher was Professor of Laryngology and Rhinology at the University of Denver Medical Department, he was a member of the staff of St. Luke's Hospital, on the consulting staff of Children's Hospital, and for two years on the Advisory Board of Presbyterian Hospital. He was a thirty-second degree Mason and a Shriner.

Dr. Gallaher was Secretary of the Section on Laryngology and Otology of the American Medical Association 1894-1895, a Fellow of the American College of Surgeons, and a member of the American Medical Society of Vienna, the American Academy of Ophthalmology and Otolaryngology, and the American Laryngological, Rhinological and Otological Society.

His sister, Miss Isabel I. Gallaher, survives him.

Dr. Frank Yuba Gilbert

Dr. Gilbert was born in Orono, Maine, on March 28, 1878, and died in Augusta, Maine, on January 18, 1937.

He graduated from the Medical School of Maine, Portland, in 1901, and served his interneship at the Butler Hospital, Providence, Rhode Island, and at the Maine Eye and Ear Infirmary.

Dr. Gilbert served during the World War, was attending specialist in diseases of the ear, nose and throat at the U. S. Marine Hospital, a member of the examining board of the U. S. Veterans' Bureau, Past Commander of the Harold T. Andrews Post of the American Legion and Lieutenant-Colonel U. S. Reserve Officers.

He was a member of the House of Delegates of the American Medical Association in 1914, 1917, 1919, 1927, and 1930, President of the Maine Medical Association in 1928, and founder and editor,

from 1910 to 1930, of the Maine Medical Journal. His recreation was hunting and fishing.

Dr. Gilbert was a member of the American Academy of Ophthalmology and Otolaryngology and the New England Ophthalmological Society.

At the time of his death he was on the staff of the Maine Eye and Ear Infirmary.

His wife and one daughter survive him.

Dr. Samuel Pellman Glover

Dr. Glover was born in Hartleton, Union County, Pennsylvania, on February 29, 1860, and died in Altoona, on October 3, 1936.

Dr. Glover received his preliminary education at the Bloomsburgh Normal School and Lafayette College, and his medical degree from the University of Pennsylvania in 1884.

He served an interneship at Blockley Hospital, Philadelphia, then spent five years with the American General Clinic at Beirut, Syria, and later did postgraduate work in Vienna and at the Wills Eye Hospital, Philadelphia. He began the practice of his specialties in Altoona, Pennsylvania, and at the time of his death was Chief of the Eye Department at Altoona Hospital, and laryngologist at Creson Sanatarium.

Dr. Glover was First Lieutenant and Assistant Surgeon to the Fifth Regiment, Pennsylvania Volunteers in the Spanish-American War.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and state medical societies.

Dr. Glover was a philatelist and an enthusiastic member of historical societies.

He is survived by his wife, one son and one daughter.

Dr. Louis David Green

Dr. Green was born in Latvia, on April 21, 1882, came to the United States at the age of two, and died in San Francisco, California, on January 14, 1937.

He graduated from Gross Medical College, Denver, Colorado, in 1909, and began the practice of Medicine in San Francisco in

1909. Later he specialized in ophthalmology and studied in Vienna in 1911, and in India in 1915 under Colonel Smith, particularly in regard to cataract.

Dr. Green served during the World War as Captain, and in 1928 opened the Green's Eye Hospital in San Francisco with his brother and nephew. He was a frequent contributor of medical articles and inventor of ophthalmological instruments.

Dr. Green was a member of the American Academy of Ophthalmology and Otolaryngology, the Pacific Coast Oto-Ophthalmological Society and the Pan-American Medical Association, and a Fellow of the American College of Surgeons.

His wife, one son and two daughters, survive him.

Dr. Matthias David Haspel

Dr. Haspel was born November 29, 1882, at Point Michel, Louisiana, and died June 26, 1937, at New Orleans.

After receiving his medical degree from Tulane University of Louisiana Medical Department in 1905, Dr. Haspel went to Vienna for two years' study in ear, nose and throat, returning to New Orleans in 1909.

At the time of his death Dr. Haspel was on the staffs of the Baptist Hospital and the Charity Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the Louisiana State Medical Society, and Phi Alpha Sigma medical fraternity.

During the World War he served as specialist for the Department of the Interior, Bureau of Pensions, and as Medical Officer on the Advisory Board.

He is survived by his wife, one son and one daughter.

Dr. Henry Louis Hilgartner

Dr. Hilgartner was born in Austin, Texas, on July 10, 1868, and died in Atlantic City, New Jersey, on June 9, 1937.

He received his medical degree from the University of Maryland Medical College in 1889, and his B.A. degree from the University of Texas in 1896. Following a period of postgraduate study in New York, Baltimore, and Vienna, he began the practice of medicine in Austin, and at the time of his death was on the staffs

of Seton Infirmary, St. David's and Brackenridge Hospitals, and oculist to the Texas Institution for Deaf, Dumb and Blind Colored Youths. At one time he was oculist to the Texas State School for the Blind.

During the World War, Dr. Hilgartner was consulting eye, ear, nose and throat surgeon and examiner for the United States School for Aeronautics.

He was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

He was fond of reading and travel.

He is survived by his wife and one son.

Dr. Eugene Eunson Hinman

Dr. Hinman was born in Albany, New York, on May 23, 1876, and died there on January 20, 1937.

He received his medical degree from the Albany Medical College in 1899, then took postgraduate work in New York City, and with Dr. Chevalier Jackson in Philadelphia. He began the practice of medicine in his home town, and at the time of his death was Clinical Professor of Otolaryngology at his alma mater, on the staffs of Albany Hospital and Child's Hospital, as well as Consultant in Otolaryngology to the Memorial Hospital of Greene County, Catskill, and St. Luke's Hospital, Newburgh.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, Past President and Secretary of the Albany County Medical Society and member of various local and state medical societies. Just before his death, Dr. Hinman had accepted an appointment as Fellow of the International College of Surgeons as head of the Department from New York State.

For many years Dr. Hinman was a Major in the Medical Department of the New York National Guard. Outside of medicine he was keenly interested in Masonry, was a Shriner, and co-author of two Masonic books. He was devoted to the service of the Grand Lodge Museum and a student in archæology, especially of Arabia and all parts of the Holy Land.

His wife and one daughter survive him.

Dr. Edward Daniel Hurley

Dr. Hurley was born October 22, 1881, in Boston, Massachusetts, and died there on June 8, 1936.

He graduated from Harvard Medical School in 1904, later took postgraduate courses in eye work in Vienna, and began the practice of his specialty in Boston in 1908.

At the time of his death Dr. Hurley was ophthalmologist-in-chief of the Carney Hospital, and consulting ophthalmologist to St. Margaret's Hospital, both of Boston, and consulting ophthalmologist to the Choate Memorial Hospital, Woburn, and St. Mary's Infant Asylum.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and member of local and state medical and special societies.

His hobbies were golf, bowling and gardening, and he was an ardent member of the Knights of Columbus.

His wife, three sons and one daughter survive him.

Dr. Arthur Frederick Jackle

Dr. Jackle was born January 16, 1882, at Dunkirk, New York, and died March 6, 1937, at Pasadena, California.

He was a graduate of the University of Buffalo School of Medicine in 1907, later did postgraduate work at the University of Michigan and the New York Postgraduate Hospital, and took up the practice of medicine in Dunkirk, New York.

Dr. Jackle was at one time on the staffs of the Buffalo General Hospital and the Sisters of Charity Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and local medical societies. He had traveled abroad extensively.

He is survived by an uncle and a nephew.

Dr. Herman Jarecky

Dr. Jarecky was born in New York City on June 14, 1863, and died there on March 14, 1937.

He received his A.B. degree from the College of the City of New York in 1883, and his medical degree from the College of Physicians and Surgeons, Medical Department of Columbia Col-

lege, in 1886, following which he was House Surgeon to the City Hospital for one year.

At one time Dr. Jarecky was Assistant Surgeon to the Manhattan Eye, Ear and Throat Hospital, and at the time of his death was Consultant Otologist and Laryngologist to Sydenham Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and various city and state medical societies, and was the author of various medical papers.

He is survived by his wife and one daughter.

Dr. John Loring Johnson

Dr. Johnson was born in Houlton, Maine, on June 7, 1888, and died in Bangor, Maine, on January 14, 1937.

He graduated from Bowdoin College with the degree of A.B. in 1911, and gained his medical degree from Columbia University College of Physicians and Surgeons in 1914. After some postgraduate work at the Manhattan Eye and Ear Infirmary, he began the practice of his specialty in Bangor.

At the time of his death, Dr. Johnson was on the staff of the Eastern Maine General Hospital, having been President of the staff for several years, and consultant in oto-rhino-laryngology to the Presque Isle (Maine) General Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Board of Otolaryngology, Fellow of the American College of Surgeons, member of the Maine Medical Association and President in 1935-36.

Dr. Johnson was a staunch Mason and active in civic interests, being a member of the Bangor city council.

He is survived by his wife and mother.

Dr. Miner Raymond Kendall

Dr. Kendall was born in Waterford, Pennsylvania, on July 3, 1888, and died in Cleveland, Ohio, July 14, 1937.

He graduated from Bucknell University in 1911 with the degree of Ph.B., M.Sc., in 1912, and received his medical degree from Western Reserve School of Medicine in 1914.

He was demonstrator in Diseases of the Ear, Nose and Throat at Western Reserve School of Medicine from 1920 to 1925, and

Instructor from 1925 to 1930. At the time of his death he was Visitant Otolaryngologist to the St. Luke's Hospital, and Consultant Otolaryngologist to the United States Marine Hospital, United States Public Health Service.

He was a member of the American Academy of Ophthalmology and Otolaryngology, member of the Board of Directors of the Cleveland Association for the Hard of Hearing, and of local and state medical societies. Golf and clay modeling were his hobbies.

He served during the World War as First Lieutenant and later as Captain at the Base Hospitals of Camp Meade, Camp Wheeler, No. 21 at Toul, France, and No. 120 at Tours, France, at the latter as Chief of the Otolaryngological Service.

His wife and mother survive him.

Dr. George Lincoln King

Dr. King was born in Berlin Center, Ohio, in 1865, and died in Alliance, Ohio, on July 8, 1937.

He was educated at Mount Union College, Alliance, received his medical degree at Cleveland Medical College in 1895, and took postgraduate work at the Philadelphia Polyclinic Hospital and the New York Eye, Ear, Nose and Throat Hospital.

Dr. King was on the staff of the Alliance City Hospital from 1900 until the time of his death, was a member of the Board of Education for six years, and a member and former President of the Stark County Medical Society.

He was a member of the American Academy of Ophthalmology and Otolaryngology. He invented the tonsil knife and was the author of numerous articles dealing with the eye, ear, nose and throat, chief among them being the use of the commercial magnet in removing steel from the eyeball. His writings appeared in many professional journals throughout the country.

His wife, two sons, and one daughter survive him.

Dr. Samuel Kirkpatrick

Dr. Kirkpatrick was born on August 19, 1866, in Cahaba, Alabama, and died January 6, 1937, at Selma, Alabama.

He was graduated from Southern University, Greensboro, Alabama, and received his medical degree in 1888 from Vanderbilt University School of Medicine, Nashville, Tennessee. He began

the practice of medicine in Selma, and later took postgraduate work at the New York Eye and Ear Hospital and the New York Polyclinic Hospital, to aid him in the specialty he had chosen.

Dr. Kirkpatrick aided in the development of the Vaughan Memorial Hospital, on the staff of which he was oculist and aurist, and for many years before his death he was oculist and aurist to the Southern Railroad and the Burwell Infirmary.

He was a Past President of the Medical Association of the State of Alabama and the Dallas County Medical Society, a member of the American Academy of Ophthalmology and Otolaryngology, and a Fellow of the American College of Surgeons. He wrote and published numerous medical papers and had extensive farming interests as well.

His wife, three daughters and three grandchildren survive him.

Dr. James William Leech

Dr. James William Leech was born in Worcester, Massachusetts, on January 22, 1881, and died at Providence, Rhode Island, on October 6, 1936.

Obtaining his early education from the Providence public schools and Brown University, he received his medical degree from the University of Pennsylvania Medical School in 1904, afterward taking postgraduate work at the Wills Eye Hospital in Philadelphia.

He began the practice of his specialty in Providence in 1907, and at the time of his death was surgeon of the Eye Department, Rhode Island Hospital; surgeon-in-chief Eye Department, Charles V. Chapin Hospital; consulting ophthalmologist to the Providence Lying-In Hospital, Providence, Memorial Hospital, Pawtucket, and Westerly (Rhode Island) Hospitals, and consulting laryngologist to the Butler Hospital.

Dr. Leech was a Fellow of the American Academy of Ophthalmology and Otolaryngology and the American College of Surgeons; a member of the New England Ophthalmological Society, member and for twenty years Secretary of the Rhode Island Medical Society, member and Past President of the Rhode Island Ophthalmological and Otological Society.

Dr. Leech contributed many articles on his specialty to the medical magazines, and was greatly interested in reading and gardening.

He is survived by his widow, one daughter and one son.

Dr. Lorenzo B. Lockard

Dr. Lockard was born in Salem, Ohio, on November 12, 1872, and died at his home in Denver, Colorado, on July 31, 1937.

He received his early education at Syracuse University, and his medical degree from the University of Pennsylvania in 1893. After taking postgraduate work in Vienna, Dr. Lockard moved to Denver in 1901 and began the practice of medicine. He achieved prominence in the management of tuberculosis of the larynx. His treatise on this subject has been accepted as authoritative, both in America and abroad, and he contributed many articles to various medical journals.

Dr. Lockard was a life member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American Laryngological Association and of the American College of Surgeons, local and state medical societies and national and international societies for the prevention of tuberculosis. Those who knew him well recall his courteous and genial personality, and his friendship will be greatly missed by the Fellows in the various associations.

During the World War, Dr. Hilgartner was consulting eye, archæology. Failing health and a desire to study caused Dr. Lockard to retire from active practice several years ago, but before his retirement he was laryngologist and rhinologist to the Jewish Consumptives' Relief Sanatorium, the Evangelical Lutheran Sanatorium, laryngologist to the National Jewish Hospital for Consumptives, member of the Board of Directors of the Agnes Memorial Sanatorium, one time Professor of Anatomy at Toledo Medical College, and on the staffs of St. Luke's Hospital and Children's Hospital.

He is survived by his wife and two sisters.

Dr. Alvin J. Lorie

Dr. Lorie was born in Kansas City, Missouri, on August 31, 1886, and died at Miami Beach, Florida, on March 8, 1937.

He received his early education in Kansas City, his A.B. degree in 1908, and his medical degree in 1911 from the University of Michigan. Dr. Lorie taught rhinology at the University of Michigan from 1911 to 1914, and was also on the teaching staff of the Western Dental College and International School of Orthodontia.

After postgraduate study at the University of Michigan, he took up the practice of ear, nose and throat in Kansas City in 1914.

Dr. Lorie was for twenty years Chief of the Ear, Nose and Throat Department of the Kansas City General Hospital. He was also Vice-President of the staff of St. Joseph Hospital, Chief of Staff at Menorah Hospital, and on the staff of Research Hospital and St. Luke's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, member and Vice-President of the Midwest Section of the American Laryngological, Rhinological and Otological Society, and a Fellow of the American College of Surgeons. He was a Past President of Phi Rho Sigma and Phi Kappa Sigma Medical Fraternities; a life trustee of the United Jewish Charities, and a member of the Jackson County and Southwest Clinical Societies.

He is survived by his wife and one son.

Dr. Francis Gabriel Miniter

Dr. Miniter was born on May 25, 1884, at Ayer, Massachusetts, and died in Boston, Massachusetts, on April 10, 1937.

He graduated from the Massachusetts College of Pharmacy in 1909, and received his medical degree from Tufts College Medical School in 1913, following which he began the practice of medicine in Boston.

Dr. Miniter was Assistant Laryngological Surgeon to the Boston City Hospital, and Chief Laryngologist at Carney Hospital and St. Margaret's Hospital in Boston, and the Cambridge City Hospital, Cambridge.

During the World War, Dr. Miniter served as United States Navy Medical Officer from 1917 to 1921.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and member of local and state medical societies.

Dr. Miniter is survived by his mother, one sister and two brothers.

Dr. Bernard Anthony O'Hora

Dr. O'Hora was born in Black Earth, Wisconsin, on August 27, 1889, and died in Detroit, Michigan, on October 29, 1936.

He received the degree of B.S. from the University of Wisconsin in 1914, and M.D. from Washington University School of Medicine in St. Louis in 1917.

Dr. O'Hora served as First Lieutenant in the Army Medical Corps for eighteen months during the World War, and was on the staff of the Henry Ford Hospital until 1925. Following postgraduate work under Dr. Mosher, in Boston, he took up the private practice of ear, nose and throat work in Detroit.

He was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

At the time of his death Dr. O'Hora was Chief of the Department of Otolaryngology of the Detroit Woman's Hospital, and on the staffs at Harper Hospital and Children's Hospital.

He was a member and Past Commander of the Business and Professional Men's Post of the American Legion.

Dr. O'Hora's wife died in 1934. He is survived by three sons, his mother, three brothers and three sisters.

Dr. George Oliver Sharrett

Dr. George Oliver Sharrett was born in Baltimore, Maryland, on April 19, 1887, and died in Cumberland, Maryland, on August 27, 1936.

Dr. Sharrett received his early education from Baltimore City College and his medical degree from Maryland University. After graduation he did postgraduate work in the Baltimore Eye, Ear, Nose and Throat Hospital, and began the practice of medicine in Baltimore in 1908.

Dr. Sharrett served during the World War, first at the Medical Officers' Training Camp at Fort Oglethorpe, Georgia, later at the Base Hospital at Camp Sheridan, Montgomery, Alabama. He was sent to Columbia University in New York City to take a special course in brain surgery, and at the outbreak of the influenza epidemic was transferred to Fort Ontario, Oswego, N. Y. He received the rank of Captain on May 30, 1918.

At the time of his death, Dr. Sharrett was consulting oculist and otolaryngologist to the Miners Hospital, Frostburg; the Hazel McGilvery Hospital, Meyersdale, Pennsylvania; the Allegheny

Hospital, and the Memorial Hospital, Cumberland, and oculist for the Western Maryland Railroad.

He was prominent in Masonry, and worked untiringly for everything that pertained to community well-being.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American Rhinological, Otological and Laryngological Society, Fellow of the American College of Surgeons, member of the State Board of Medical Examiners, and Past President of the Medical and Chirurgical Faculty of Maryland and the Allegheny-Garrett Counties Medical Society.

He is survived by his wife, one son, his parents and a brother.

Dr. Elbert Byron Swerdfeger

Dr. Swerdfeger was born on June 25, 1876, at Sabetha, Kansas, and died on February 9, 1937, at Denver, Colorado.

He received his medical degree from Denver Homeopathic College in 1904, and practiced his specialties in that city, after taking postgraduate work at the Harvard Medical School and the University of Pennsylvania.

Dr. Swerdfeger was Past President and Vice-President of the State Board of Medical Examiners, Assistant Professor of Otolaryngology at the University of Colorado School of Medicine, member of the American Academy of Ophthalmology and Otolaryngology, and Fellow of the American College of Surgeons. He served during the World War.

At the time of his death, Dr. Swerdfeger was otolaryngologist to the Children's, Denver General, Colorado General and St. Luke's Hospitals, Colorado Christian Home, the State Home for Dependent Children, and the Presbyterian Hospital.

Dr. Swerdfeger was greatly interested in the Masonic Orders, being a member of the Consistory and Shrine.

He is survived by his wife, two sons and a daughter.

Dr. Milton Thomashefsky

Dr. Thomashefsky, of Brooklyn, New York, died on May 15, 1937, at the age of thirty-eight.

He received his medical degree from Columbia University College of Physicians and Surgeons, New York, in 1921, and at the

time of his death was Associate in the Ear, Nose and Throat Departments of the Jewish and Greenpoint Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, a Fellow of the American College of Surgeons, and member of the Medical Society of the State of New York.

Dr. Charles Christian Walker

Dr. Walker was born September 24, 1875, at Cleveland, Ohio, and died in Detroit, Michigan, on September 28, 1936.

He was educated at Oberlin Preparatory School and College, obtained his medical degree from the University of Michigan in 1904, and then took two years of postgraduate work at the University of Pennsylvania. Later he worked under Dr. Harvey Howard, in Canton, China, and with Lieutenant-Colonel Henry Smith of the Indian Medical Service, Amritsar, India.

Dr. Walker was Professor of Diseases of the Eye at Bangkok Royal Medical College, and for years conducted his own private hospital of fifty beds for eye diseases in Bangkok, Siam. He learned the Siamese language and served the United States Legation as medical adviser and the Standard Oil Company as medical attendant. His little hospital in Siam grew to be a huge clinic, ministering to the needs of a city of a million people.

At the time of his death, Dr. Walker was on the staffs of the Harper Hospital and the Children's Free Hospital in Detroit.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

His wife, two sons and a daughter survive him.

Dr. Charles James Wetherbee

Dr. Wetherbee was born on a farm in Porter Township, Cass County, Michigan, on July 19, 1868, and died at Twin Falls, Idaho, on February 9, 1937.

He received his medical degree from the Detroit College of Medicine in 1901, after having attended the University of Michigan, and served his interneship at the Harper Hospital in Detroit. Later he was Assistant Superintendent of the Solvay Hospital at Delray, Michigan, for one year, and then took up the practice of

medicine at Jones, Michigan. Desiring to specialize in eye, ear, nose and throat practice, Dr. Wetherbee took postgraduate courses in New York City, Chicago, Denver, Rochester (Minnesota), San Francisco, and Seattle, and settled in Twin Falls in 1918.

Dr. Wetherbee was greatly interested in civic problems, being at one time Mayor of Buhl, Idaho. He also enjoyed sports of the field and stream, and was a member of the Masonic Order, serving one year as Master of the Masonic Lodge, and was also a member of the Royal Astronomical Society of Canada.

At the time of his death, Dr. Wetherbee was on the staff of the Twin Falls County General Hospital. He was a member of the American Academy of Ophthalmology and Otolaryngology, the Pacific Coast Oto-Ophthalmological Society, and a Fellow of the American College of Surgeons.

His wife, a stepdaughter, and one granddaughter survive him.

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of the microfilm user.